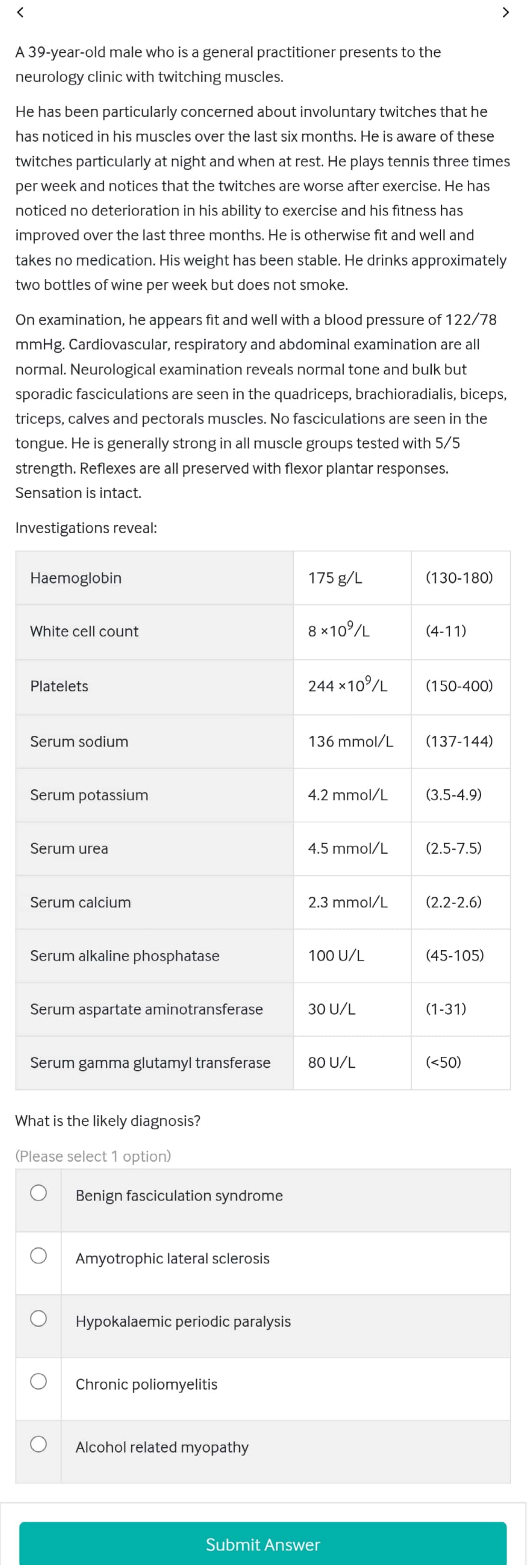


A 39-year-old male who is a general practitioner presents to the neurology clinic with twitching muscles.

He has been particularly concerned about involuntary twitches that he has noticed in his muscles over the last six months. He is aware of these twitches particularly at night and when at rest. He plays tennis three times per week and notices that the twitches are worse after exercise. He has noticed no deterioration in his ability to exercise and his fitness has improved over the last three months. He is otherwise fit and well and takes no medication. His weight has been stable. He drinks approximately two bottles of wine per week but does not smoke.

On examination, he appears fit and well with a blood pressure of 122/78 mmHg. Cardiovascular, respiratory and abdominal examination are all normal. Neurological examination reveals normal tone and bulk but sporadic fasciculations are seen in the quadriceps, brachioradialis, biceps, triceps, calves and pectorals muscles. No fasciculations are seen in the tongue. He is generally strong in all muscle groups tested with 5/5 strength. Reflexes are all preserved with flexor plantar responses. Sensation is intact.

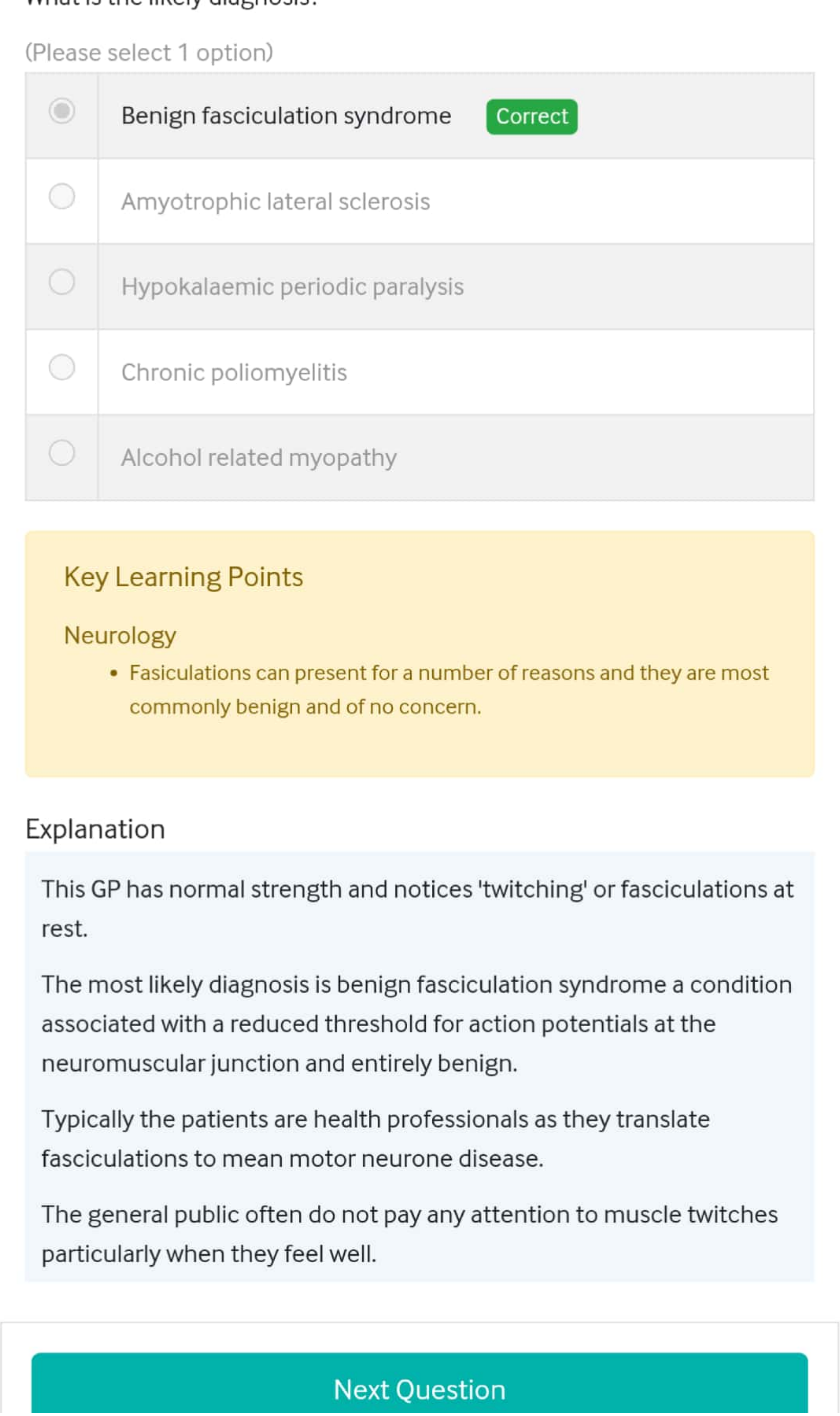
Investigations reveal:

Haemoglobin	175 g/L	(130-180)
White cell count	8 ×10^9/L	(4-11)
Platelets	244 ×10^9/L	(150-400)
Serum sodium	136 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	4.5 mmol/L	(2.5-7.5)
Serum calcium	2.3 mmol/L	(2.2-2.6)
Serum alkaline phosphatase	100 U/L	(45-105)
Serum aspartate aminotransferase	30 U/L	(1-31)
Serum gamma glutamyl transferase	80 U/L	(<50)

What is the likely diagnosis?

(Please select 1 option)

- ○ Benign fasciculation syndrome
- ○ Amyotrophic lateral sclerosis
- ○ Hypokalaemic periodic paralysis
- ○ Chronic poliomyelitis
- ○ Alcohol related myopathy



What is the likely diagnosis?

(Please select 1 option)

● Benign fasciculation syndrome **Correct**

○ Amyotrophic lateral sclerosis

○ Hypokalaemic periodic paralysis

○ Chronic poliomyelitis

○ Alcohol related myopathy

Key Learning Points

Neurology

- Fasiculations can present for a number of reasons and they are most commonly benign and of no concern.

Explanation

This GP has normal strength and notices 'twitching' or fasciculations at rest.

The most likely diagnosis is benign fasciculation syndrome a condition associated with a reduced threshold for action potentials at the neuromuscular junction and entirely benign.

Typically the patients are health professionals as they translate fasciculations to mean motor neurone disease.

The general public often do not pay any attention to muscle twitches particularly when they feel well.

Question 2

Question 2 of 129

‹ ›

An 85-year-old lady is admitted from a nursing home with reduced GCS of 12. She had been complaining of a headache for two days prior to admission and had become increasingly confused.

On admission, she is pyrexial and has a witnessed tonic-clonic seizure. Examination of her cardiorespiratory system is unremarkable but she has evidence of neck stiffness and up-going planters.

CT head is normal.

Lumbar puncture shows an elevated CSF opening pressure.

CSF count is predominantly lymphocytes and Gram stain shows Gram-positive bacilli.

What is the most likely organism?

(Please select 1 option)

- ○ *Neisseria meningitides*
- ○ *Haemophilus influenzae*
- ○ *Streptococcus pneumoniae*
- ○ *Listeria monocytogenes*
- ○ *Mycobacterium tuberculosis*

○ *Streptococcus pneumoniae*

○ *Listeria monocytogenes* This is the correct answer

○ *Mycobacterium tuberculosis*

Key Learning Points

Neurology

- Listeria monocytogenes is a Gram-positive bacillus which affects immunosuppressed patients, or patients at extremes of age.

Explanation

Listeria monocytogenes is a Gram-positive bacillus which affects immunosuppressed patients, or patients at extremes of age. It can present with reduced conscious level, seizures, and cranial nerve palsies. Cerebrospinal fluid (CSF) shows predominantly a lymphocytosis with a normal glucose.

Bacterial meningitis caused by *Haemophilus influenzae, Neisseria meningitides* and *Streptococcus pneumoniae* has CSF which shows a reduced glucose and predominantly polymorphs.

Haemophilus influenzae is a Gram-negative bacillus.

Neisseria meningitides is a Gram-negative coccus.

Streptococcus pneumoniae is a Gram-positive coccus.

Mycobacterium tuberculosis has CSF which shows predominantly lymphocytes but the history is sub-acute onset and the organism is an acid and alcohol fast bacillus on Ziehl-Neelsen staining.

Next Question

A 65-year-old woman was admitted to a gynaecology ward for repair of a cervical prolapse. She had an uneventful operation and was up and about within two days. On the fifth postoperative day she was found wandering the adjacent ward in a confused state.

She had been complaining of a throbbing headache within the preceding hour but had otherwise been well in herself. The period of confusion lasted approximately two hours during which she would repeatedly ask, 'What am I doing here?', otherwise she was able to converse appropriately.

Following the episode she had no recollection of what had happened and was able to resume a normal conversation although needed to be reminded how long she had been in hospital.

She had a past medical history of hypertension, hyperlipidaemia and migraine. She took regular atenolol and simvastatin. She was a non-smoker and did not drink any alcohol. There was no family history of medical problems.

On examination she complained of headache. Her vital signs were all normal. Cranial nerve examination was normal and there was no evidence of nystagmus. Peripheral nervous system examination including plantar reflex responses was normal. Chest and cardiovascular examination were entirely normal.

Investigations revealed:

Haemoglobin	112 g/L	(130-180)
White cell count	11.3 $\times 10^9$/L	(4-11)
Platelets	245 $\times 10^9$/L	(150-400)
ESR (Westergren)	18 mm/1st hour	(0-15)
Serum sodium	136 mmol/L	(137-144)
Serum potassium	4.5 mmol/L	(3.5-4.9)
Serum urea	4.2 mmol/L	(2.5-7.5)
Serum creatinine	89 µmol/L	(60-110)
Serum C reactive protein	15 IU/L	(<5)
Urinalysis	Protein ++	
	Blood +	

Given the clinical account and investigations so far, what is the most likely diagnosis in this patient?

(Please select 1 option)

○ Transient global amnesia

○ Vertebrobasilar migraine

○ Carotid artery territory transient ischaemic attack (TIA)

○ Temporal lobes seizures

○ Delirium

Submit Answer

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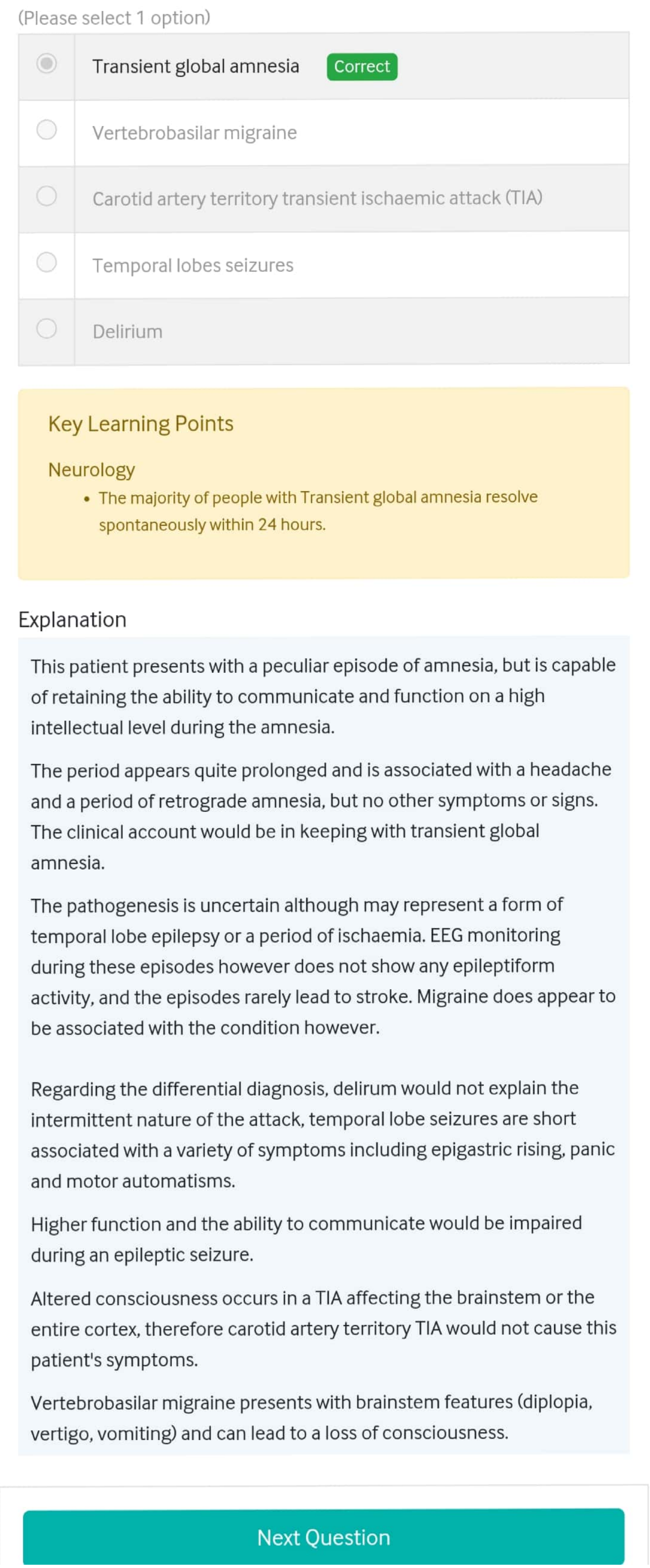


(Please select 1 option)

● Transient global amnesia Correct

○ Vertebrobasilar migraine

○ Carotid artery territory transient ischaemic attack (TIA)

○ Temporal lobes seizures

○ Delirium

Key Learning Points

Neurology

- The majority of people with Transient global amnesia resolve spontaneously within 24 hours.

Explanation

This patient presents with a peculiar episode of amnesia, but is capable of retaining the ability to communicate and function on a high intellectual level during the amnesia.

The period appears quite prolonged and is associated with a headache and a period of retrograde amnesia, but no other symptoms or signs. The clinical account would be in keeping with transient global amnesia.

The pathogenesis is uncertain although may represent a form of temporal lobe epilepsy or a period of ischaemia. EEG monitoring during these episodes however does not show any epileptiform activity, and the episodes rarely lead to stroke. Migraine does appear to be associated with the condition however.

Regarding the differential diagnosis, delirum would not explain the intermittent nature of the attack, temporal lobe seizures are short associated with a variety of symptoms including epigastric rising, panic and motor automatisms.

Higher function and the ability to communicate would be impaired during an epileptic seizure.

Altered consciousness occurs in a TIA affecting the brainstem or the entire cortex, therefore carotid artery territory TIA would not cause this patient's symptoms.

Vertebrobasilar migraine presents with brainstem features (diplopia, vertigo, vomiting) and can lead to a loss of consciousness.

‹ ›

A 17-year-old woman presented to her GP with poor co-ordination, falls and weight loss. She gave an eight month history of unsteadiness of gait and more recently she had developed slurred speech and a tremor of the upper limb. She denied any alteration in bowel habit, but commented that she had had increased urinary frequency.

She had been referred by her GP to the cardiologists with recurrent chest pain and palpitations and was awaiting an appointment. She had no known past medical history, but had been labelled clumsy as a child. There was no apparent family history of neurological disease.

On examination she was alert and orientated. Cranial nerve examination revealed scanning speech and gaze-evoked horizontal nystagmus.

On examination of the upper limb, there was a bilateral intention tremor with marked past pointing. Tone was reduced at both wrists and both supinator reflexes were absent. Light touch was felt at the elbow bilaterally.

On examination of the lower limb there was bilateral pes cavus. Tone was markedly reduced at both ankles and there was pyramidal weakness of both legs with absent knee and ankle reflexes and extensor plantar responses. She had a high steppage gait and Romberg's test was positive. On cardiovascular examination there was an ejection systolic murmur along the left sternal edge.

Investigations showed:

Haemoglobin	142 g/L	(130-180)
Mean cell volume	92 fL	(80-96)
White cell count	5.08 $\times 10^9$/L	(4-11)
Platelets	276 $\times 10^9$/L	(150-400)
Serum sodium	138 mmol/L	(137-144)
Serum potassium	3.9 mmol/L	(3.5-4.9)
Serum urea	3.1 mmol/L	(2.5-7.5)
Serum creatinine	75 μmol/L	(60-110)
Serum C reactive protein	2 U/L	(<5)
Fasting plasma glucose	10.2 mmol/L	(3-6)
Serum folate	9.5 μg/L	(2-11)
Serum B_{12}	601 ng/L	(160-760)

A lumbar puncture was performed and yielded the following data:

Opening pressure	12 cmH_2O	(5-18)
CSF protein	0.38 g/L	(0.15 - 0.45)
CSF white cell count	2 cells/ml	(<5)
CSF red cell count	1 cells/ml	(<5)
CSF lactate	1.5 mmol/L	(1-2)
CSF	Oligoclonal bands	Negative

Nerve conduction studies showed an axonal sensory motor neuropathy. The electrocardiogram showed left ventricular hypertrophy.

Given the above history and findings so far, what is the likely diagnosis?

(Please select 1 option)

- ○ Multiple sclerosis
- ○ Autosomal dominant spinocerebellar ataxia (SCA)
- ○ Mitochondrial disorder
- ○ Friedreich's ataxia
- ○ Subacute combined degeneration of the cord

Submit Answer

● Friedreich's ataxia Correct

○ Subacute combined degeneration of the cord

Key Learning Points

Genetics, Neurology

- Friedreich's ataxia is an autosomal recessive condition which affects the posterior columns, corticospinal and spinocerebellar tracts.

Explanation

This patient has all the cardinal features of Friedreich's ataxia.

Friedreich's ataxia is an autosomal recessive condition normally presenting within the first decade. The posterior columns, corticospinal and spinocerebellar tracts are affected leading to cerebellum dysfunction, spastic paraparesis and absent reflexes in lower limbs.

There is also myocardial muscle involvement leading to hypertrophy and heart failure. Bilateral pes cavus and kyphoscoliosis are common and some patients develop diabetes and optic atrophy.

An autosomal dominant SCA is a differential diagnosis for patients presenting with cerebellar dysfunction, however, one would probably expect a family history of neurological disease.

Similarly, mitochondrial disorders are a differential diagnosis of patients presenting with ataxia and systemic complications, however, absence of elevated cerebrospinal fluid (CSF) lactate would make this diagnosis unlikely.

Multiple sclerosis could account for some of the clinical features, but would not explain diabetes and peripheral sensory motor neuropathic changes.

In the presence of normal B_{12} levels, sub-acute combined degeneration of the cord is unlikely.

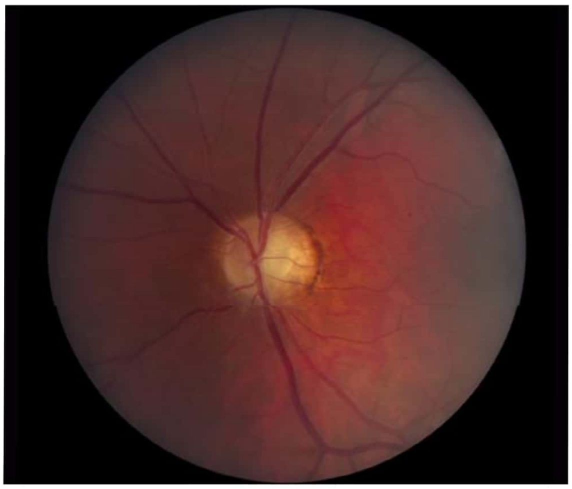
A 35-year-old woman was referred to a neurology clinic having developed recent onset unsteadiness and falls.

She had noticed a dragging of her left leg and that she appeared to be leaning to the left whilst walking. Recently she had noticed blurred vision and thought it may be due to her glasses. She had attended the opticians who had subsequently referred her to an ophthalmologist. She had seen her GP several months ago with left retro-orbital pain which subsided. She did not take any regular medication. She was a smoker of 40 cigarettes per day, and drank 25 units of alcohol per week. She had no family history of medical problems.

On examination she was alert and orientated. Her blood pressure was 135/75 mmHg, pulse was 70/min and regular and temperature was 36.7°C. She had horizontal nystagmus on looking to the left with restricted adduction of the right eye. Visual acuity was 6/9 in both eyes. The rest of the cranial nerves were normal.

On examination of the upper limb, tone, power, reflexes and sensation were all normal. There was some mild past-pointing on the left. Lower limb examination revealed increased tone on the left with brisk reflexes and a left extensor plantar response. There was mild left-sided pyramidal weakness. She walked with a broad based ataxic gait and appeared to be dragging her left leg.

The fundoscopic appearance is shown below:



Investigations revealed: Lumbar puncture:

Opening pressure	150 mm H_20	(50-180)
CSF protein	0.67 g/L	(0.15-0.45)
CSF white cell count	4 per ml	(<5)
CSF red cell count	3 per ml	(<5)
CSF oligoclonal bands	Positive	-
Serum oligoclonal bands	Negative	-
CSF lactate	2 mmol/L	(1-2)

Given the above history, fundoscopy and lumbar puncture findings, which is the most likely diagnosis?

(Please select 1 option)

- ○ Multiple sclerosis
- ○ Mitochondrial disease
- ○ Toxic amblyopia
- ○ Leber's optic neuropathy
- ○ Glaucoma

Submit Answer

● Multiple sclerosis **Correct**

○ Mitochondrial disease

○ Toxic amblyopia

○ Leber’s optic neuropathy

○ Glaucoma

Key Learning Points

Neurology

- History of optic neuritis and cerebellar/pyramidal signs in a young patient should prompt investigation for MS.

Explanation

The fundal image demonstrates a pale, clearly delineated disc. This is typical of optic atrophy.

The three leading causes of optic atrophy are:

- Multiple sclerosis
- Compression of the optic nerve, and
- Glaucoma.

Given the history of probable optic neuritis and cerebellar/pyramidal signs in a young patient, the most likely diagnosis is multiple sclerosis.

There are no features in the history to suggest an underlying mitochondrial disorder (migraine, glucose intolerance, short stature, proximal myopathy or epilepsy) and the cerebrospinal fluid (CSF) lactate is within the normal range.

Toxic amblyopia is a rare condition, secondary to excessive tobacco or alcohol consumption. The true cause is thought to be nutritional consequences of alcohol abuse as opposed to a direct toxic effect, however clearly in this case there are additional hard neurological signs and oligoclonal bands in the CSF sample.

Leber's optic atrophy is a mitochondrial disorder leading to painless visual loss typically in young males aged in their 30s. Adult-onset cases can present with an ataxic syndrome or spastic paraparesis, however given the lack of family history and normal lactate, this is less likely than multiple sclerosis.

Next Question

A 70-year-old man was referred to the general medicine outpatient clinic with short term memory loss and reduced mobility.

He was accompanied by his wife, who reported a seven month history of poor concentration and described episodes in which her husband would be in a world of his own followed by periods where he could be completely normal. More recently he had had disturbing visual hallucinations: in one episode he had seen his dead mother on several occasions in the house.

The patient's wife also reported difficulties with his mobility for approximately twelve months. He was much slower at walking and his stride had shortened; on occasions she said that he shuffled.

He had a past medical history of urinary incontinence thought secondary to prostatic hypertrophy. He was a non-smoker and drank six units of alcohol a week.

On examination he was alert but easily distractable. He scored 21/30 on mini-mental state examination, with deficiencies in visuospatial tasks, and frontal executive tasks.

His blood pressure was 138/62 mmHg lying and 134/60 mmHg standing, and pulse 67/min and regular. He had a marked dysarthria and perseveration. Cranial nerve examination was normal, although he had slightly reduced facial expression. On upper limb examination he had bilateral rigidity and bradykinesia but no rest tremor. On examination of the lower limb there was marked rigidity and he appeared to walk with a slow festinant gait with poor turning and a reduced arm swing.

Investigations revealed:

Haemoglobin	145 g/L	(130 - 180)
MCV	94 fL	(80 - 96)
MCH	30 pg	(28 - 32)
White cell count	7.9 ×10^9/L	(4 - 11)
Platelets	210 ×10^9/L	(150 - 400)
Serum sodium	137 mmol/L	(137 - 144)
Serum potassium	4.1 mmol/L	(3.5 - 4.9)
Serum urea	6.3 mmol/L	(2.5 - 7.5)
Serum creatinine	97 μmol/L	(60 - 110)
Plasma glucose	5.6 mmol/L	(3 - 6)
Serum cholesterol	5.1 mmol/L	(<5.2)

A twelve lead ECG showed sinus rhythm with a ventricular rate of 67 beats per minute.

Given the history and above findings, what is the most likely diagnosis?

(Please select 1 option)

- ○ Alzheimer's disease
- ○ Normal pressure hydrocephalus
- ○ Idiopathic Parkinson's disease with dementia
- ○ Vascular Parkinsonism
- ○ Dementia with Lewy bodies

Submit Answer

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○ Dementia with Lewy bodies **Correct**

Key Learning Points

Neurology

- Lewy body dementia classically involves complex visual hallucinations, Parkinsonism and a subcortical pattern of dementia.

Explanation

This patient has a triad of:

- Subcortical pattern of dementia
- Complex visual hallucinations, and
- Parkinsonism.

Diagnostically if dementia occurs within 12 months of the onset of parkinsonian features the patient has Lewy body disease.

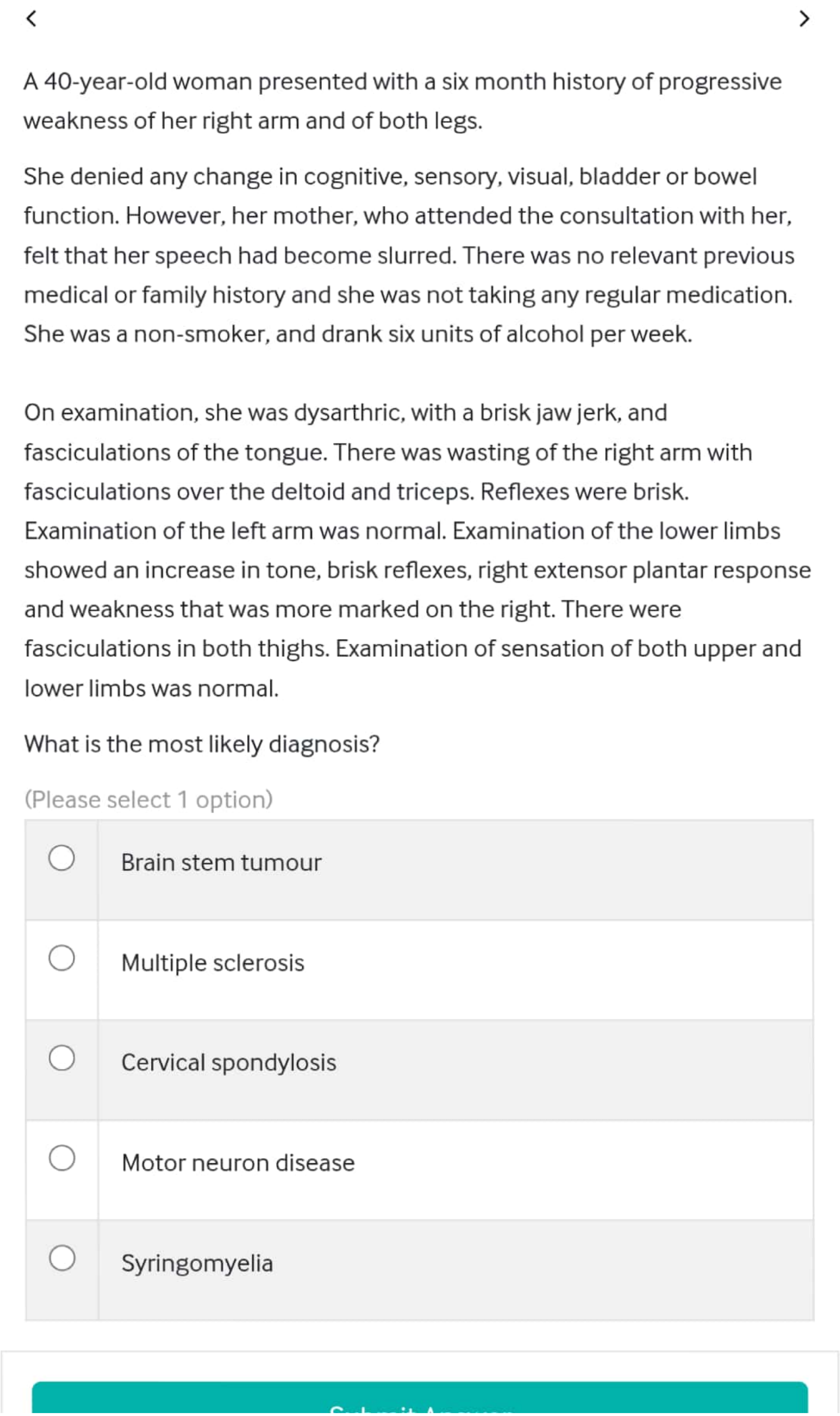
Idiopathic Parkinson's disease is often asymmetrical and normally with resting tremor. Visual hallucinations and dementia occur later in the disease process.

Vascular parkinsonism causes a lower body parkinsonism with marked rigidity and festinant gait and there are no risk factors for cerebrovascular disease in this patient.

Alzheimer's disease causes a cortical dementia with apraxias and agnosia and marked amnesia.

Normal pressure hydrocephalus causes a triad of gait apraxia, subcortical dementia and urinary dysfunction.

The gait abnormalities in the case are clearly extrapyramidal.



A 40-year-old woman presented with a six month history of progressive weakness of her right arm and of both legs.

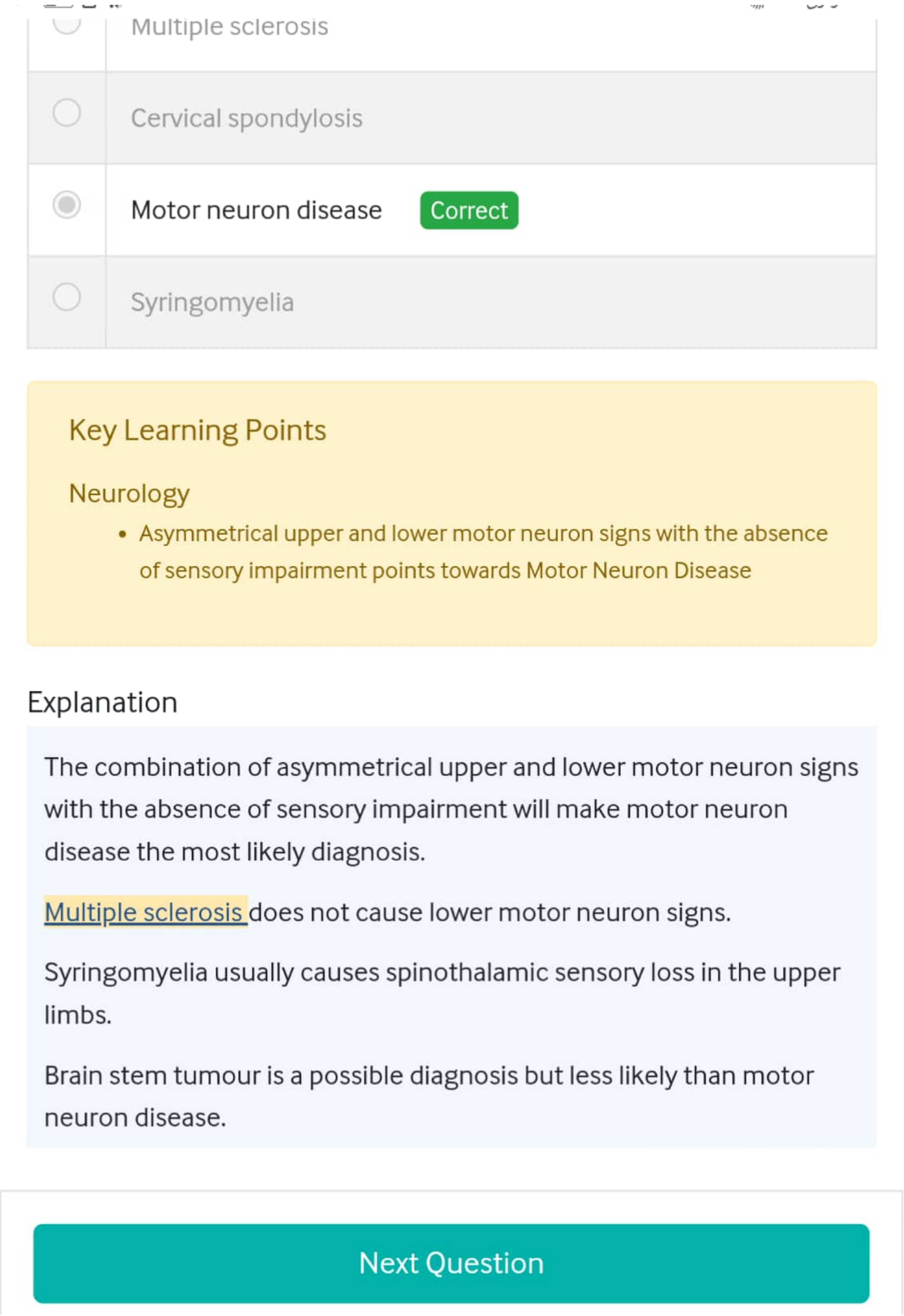
She denied any change in cognitive, sensory, visual, bladder or bowel function. However, her mother, who attended the consultation with her, felt that her speech had become slurred. There was no relevant previous medical or family history and she was not taking any regular medication. She was a non-smoker, and drank six units of alcohol per week.

On examination, she was dysarthric, with a brisk jaw jerk, and fasciculations of the tongue. There was wasting of the right arm with fasciculations over the deltoid and triceps. Reflexes were brisk. Examination of the left arm was normal. Examination of the lower limbs showed an increase in tone, brisk reflexes, right extensor plantar response and weakness that was more marked on the right. There were fasciculations in both thighs. Examination of sensation of both upper and lower limbs was normal.

What is the most likely diagnosis?

(Please select 1 option)

○ Brain stem tumour
○ Multiple sclerosis
○ Cervical spondylosis
○ Motor neuron disease
○ Syringomyelia



○ Multiple sclerosis

○ Cervical spondylosis

● Motor neuron disease **Correct**

○ Syringomyelia

Key Learning Points

Neurology

- Asymmetrical upper and lower motor neuron signs with the absence of sensory impairment points towards Motor Neuron Disease

Explanation

The combination of asymmetrical upper and lower motor neuron signs with the absence of sensory impairment will make motor neuron disease the most likely diagnosis.

Multiple sclerosis does not cause lower motor neuron signs.

Syringomyelia usually causes spinothalamic sensory loss in the upper limbs.

Brain stem tumour is a possible diagnosis but less likely than motor neuron disease.

A 41-year-old Asian man was admitted to hospital with three generalised seizures. Prior to admission he had complained of generalised headache, polydipsia and polyuria and had developed sudden onset weakness affecting his right arm and leg. In the last week he had become increasingly confused and was sleeping throughout the day.

He had a past medical history of hypertension treated with bendroflumethiazide and had recently seen his GP complaining of arthritic pain affecting his back and knees. He smoked 30 cigarettes per day.

He appeared confused and dehydrated with decreased skin turgor and dry mucous membranes. His blood pressure was 110/70 mmHg; pulse was 89/min and temperature 37.5°C. He had a left lower motor neurone (LMN) facial nerve palsy and both eyes appeared red and inflamed. Fundoscopy and the remaining cranial nerves were all normal.

Peripheral nervous system examination revealed a 4/5 right-sided hemiparesis with right extensor plantar response and brisk right knee and ankle reflexes. The left leg had normal tone however the left knee and ankle jerk appeared depressed and the left plantar response was mute.

Investigations revealed:

Haemoglobin	132 g/L	(130-180)
White cell count	9.0 $\times 10^9$/L	(4-11)
Platelets	390 $\times 10^9$/L	(150-400)
Serum sodium	159 mmol/L	(137-144)
Serum potassium	3.5 mmol/L	(3.5-4.9)
Serum urea	28.5 mmol/L	(2.5-7.5)
Serum creatinine	250 μmol/L	(60-110)
Serum calcium	3.8 mmol/L	(2.2-2.6)
Serum glucose	5.5 mmol/L	(3.0-6.0)
Plasma osmolality	455 mOsmol/kg	(278-305)
Urine osmolality	255 mOsmol/kg	(350-1000)
cANCA	Negative	

A CT scan of the brain showed a left anterior circulation infarct. A lumbar puncture was performed and CSF analysis showed:

Opening pressure	160 mmH_2O	(50-180)
CSF protein	2.7 g/L	(0.15-0.45)
CSF white cell count	160 cells per ml	≤5
	90% lymphocytes	(60-70)
CSF red cell count	5 cells per ml	(≤5)
CSF glucose	3.7 mmol/L	(3.3-4.4)
CSF culture	Negative	-
CSF cytology	Negative	-
CSF oligoclonal bands	Positive	-
Serum oligoclonal bands	Negative	-
CSF angiotensin converting enzyme	Normal	-

The chest x ray showed bilateral hilar lymphadenopathy and fibrotic interstitium.

What is the likely diagnosis in this patient?

(Please select 1 option)

○ Granulomatosis with polyangiitis

○ Tuberculous meningitis

○ Small cell lung carcinoma and lepto-meningeal involvement

○ Cerebral lymphoma

○ Neurosarcoidosis

Submit Answer

● Neurosarcoidosis **Correct**

Key Learning Points

Neurology

- Cerebrospinal fluid (CSF) analysis in neurosarcoidosis shows intrathecal IgG synthesis and a lymphocytosis

Explanation

This patient has a complex history and paraclinical findings.

He has had a left anterior circulation infarct, a left LMN facial nerve palsy and a possible neuropathy of the left lower limb. In addition he has bilateral iritis, hypercalcaemia, hypernatraemia with evidence for diabetes insipidus. Cerebrospinal fluid (CSF) analysis shows intrathecal IgG synthesis and a lymphocytosis. His CXR shows bilateral fibrosis and lymphadenopathy. These findings are all in keeping with neurosarcoidosis.

Tuberculosis meningitis rarely presents with cranial neuropathies and focal signs and is more likely to be associated with a syndrome of inappropriate antidiuretic hormone (SIADH). The CSF analysis invariably shows a markedly reduced glucose level.

In granulomatosis with polyangiitis there are often upper respiratory tract symptoms but some patients can present with mononeuropathies or vasculitic stroke. cANCA however is positive in 90% of cases.

Small cell neoplasm with meningeal spread would not cause intrathecal oligoclonal band production, and one would probably see positive cytology.

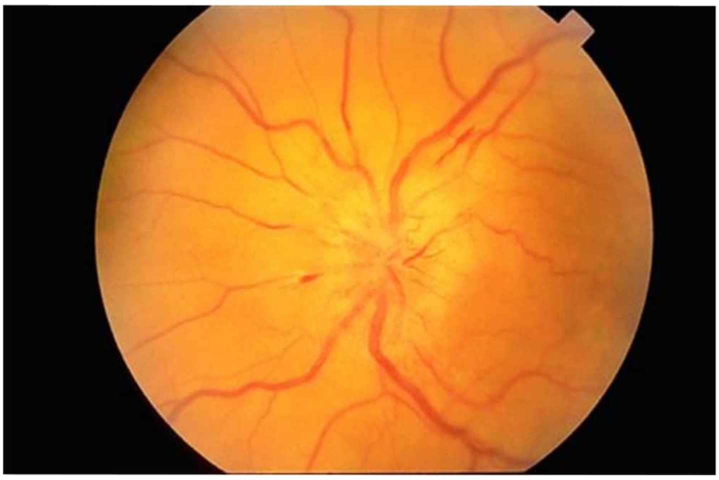
Cerebral lymphoma can present with focal neurology, cranial neuropathy and peripheral neuropathy but low glucose on CSF analysis and positive cytology.

A 23-year-old obese woman presented to the Emergency department with a four day history of progressively worsening generalised headache associated with a buzzing in her ears.

In the last few days she had noticed some blurring of her vision and reduction in her visual field. She had started to feel nauseated particularly early in the morning and on the morning of admission had vomited several times. She had a history of severe acne which was treated with long-term oral doxycycline and smoked 35 cigarettes a day.

On examination she was orientated with a Glasgow coma scale of 15/15. Visual acuity was recorded as 6/18 in both eyes. There was reduction in peripheral visual fields bilaterally and enlargement of the blind spot bilaterally. The rest of the neurological examination was entirely normal.

The fundoscopic appearance is shown below:



An MRI scan of the brain was normal.

A lumbar puncture was performed and yielded the following data:

Opening pressure	33 cm H_2O	(6-18)
CSF protein	0.42 g/L	(0.15-0.45)
CSF white cell count	2 cells per mL	(<5)
CSF red cell count	2 cells per mL	(<5)
CSF oligoclonal bands	Negative	-

Given the above clinical account, what is the likely cause for this patient's visual disturbance?

(Please select 1 option)

○	Pseudopapilloedema
○	Optic papillitis
○	Optic nerve meningioma
○	Central retinal vein occlusion
○	Idiopathic intracranial hypertension

Submit Answer

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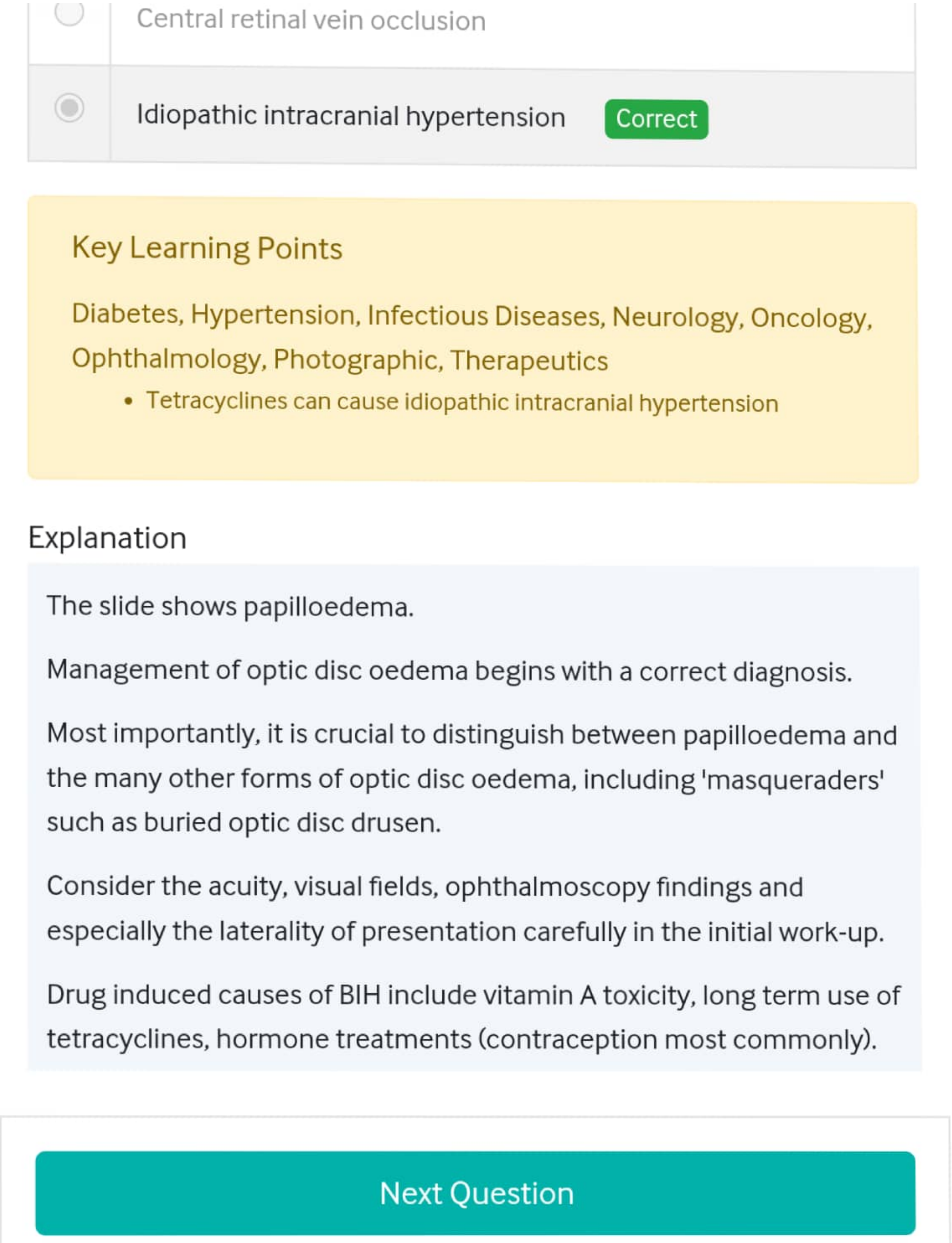


○ Central retinal vein occlusion

● Idiopathic intracranial hypertension **Correct**

Key Learning Points

Diabetes, Hypertension, Infectious Diseases, Neurology, Oncology, Ophthalmology, Photographic, Therapeutics

- Tetracyclines can cause idiopathic intracranial hypertension

Explanation

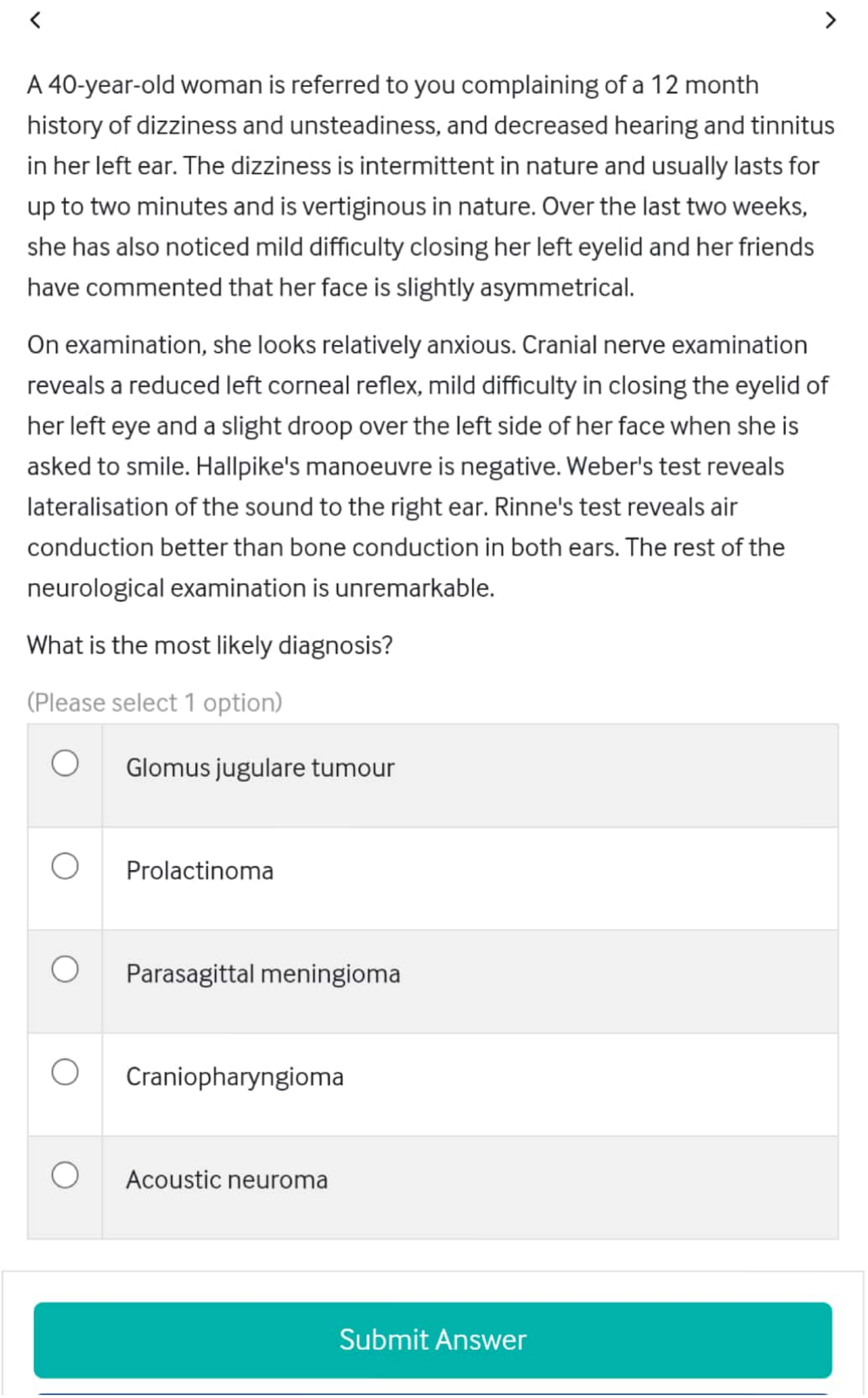
The slide shows papilloedema.

Management of optic disc oedema begins with a correct diagnosis.

Most importantly, it is crucial to distinguish between papilloedema and the many other forms of optic disc oedema, including 'masqueraders' such as buried optic disc drusen.

Consider the acuity, visual fields, ophthalmoscopy findings and especially the laterality of presentation carefully in the initial work-up.

Drug induced causes of BIH include vitamin A toxicity, long term use of tetracyclines, hormone treatments (contraception most commonly).



A 40-year-old woman is referred to you complaining of a 12 month history of dizziness and unsteadiness, and decreased hearing and tinnitus in her left ear. The dizziness is intermittent in nature and usually lasts for up to two minutes and is vertiginous in nature. Over the last two weeks, she has also noticed mild difficulty closing her left eyelid and her friends have commented that her face is slightly asymmetrical.

On examination, she looks relatively anxious. Cranial nerve examination reveals a reduced left corneal reflex, mild difficulty in closing the eyelid of her left eye and a slight droop over the left side of her face when she is asked to smile. Hallpike's manoeuvre is negative. Weber's test reveals lateralisation of the sound to the right ear. Rinne's test reveals air conduction better than bone conduction in both ears. The rest of the neurological examination is unremarkable.

What is the most likely diagnosis?

(Please select 1 option)

○ Glomus jugulare tumour

○ Prolactinoma

○ Parasagittal meningioma

○ Craniopharyngioma

○ Acoustic neuroma

○ Craniopharyngioma

● Acoustic neuroma **Correct**

Key Learning Points

Neurology

- Acoustic neuromas are the commonest cerebellopontine angle tumour.

Explanation

The combination of sensorineural deafness, facial nerve palsy and cranial nerve V involvement suggests a cerebellopontine angle tumour, for example, acoustic neuroma.

Craniopharyngiomas are more common in children than in adults. Patients usually present with signs of local compression, especially of the visual system, or hypothalamic dysfunction, for example diabetes insipidus or growth failure.

Glomus jugulare tumours tend to present with pulsatile tinnitus and conductive deafness. Cranial nerves IX, X and XI which run through the jugular foramen are also commonly affected to varying degrees. Cranial nerves VII and XII may be affected if the tumour enlarges sufficiently.

Parasagittal meningiomas may produce seizures or be entirely asymptomatic, often growing to enormous size before they are discovered. Lesions that reach sufficient size may cause spastic paraparesis and incontinence.

Prolactinomas commonly present with amenorrhoea and galactorrhoea in women and impotence in men. They are also associated with visual field defects.

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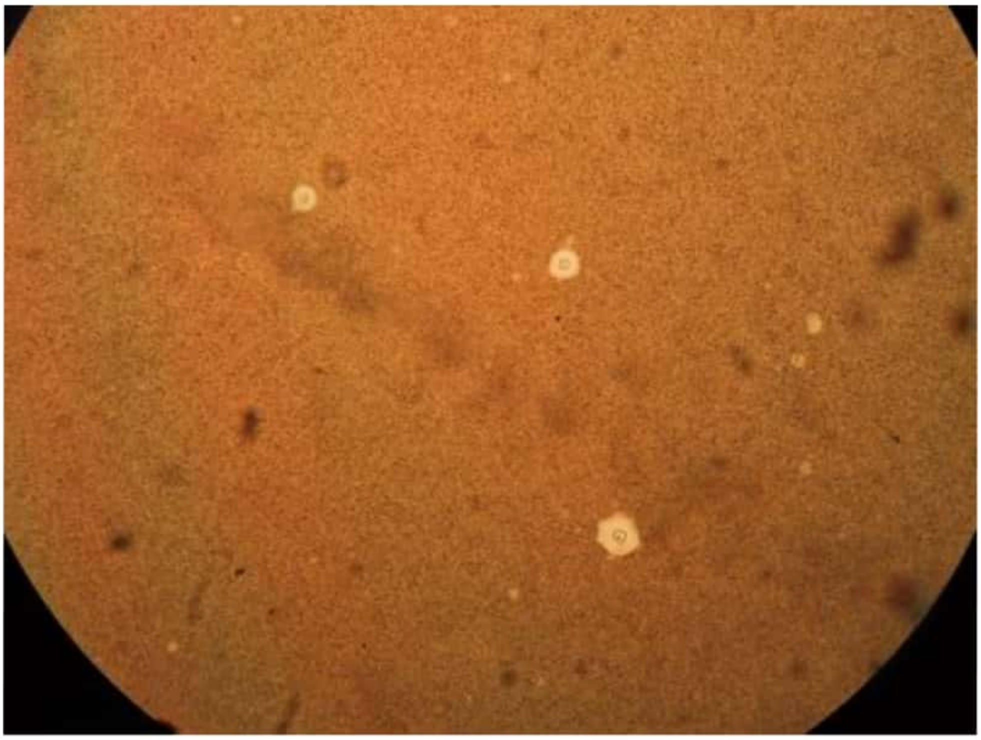
A 42-year-old Zimbabwean man was admitted to the emergency department after becoming unconscious at Heathrow airport shortly after his arrival in the United Kingdom.

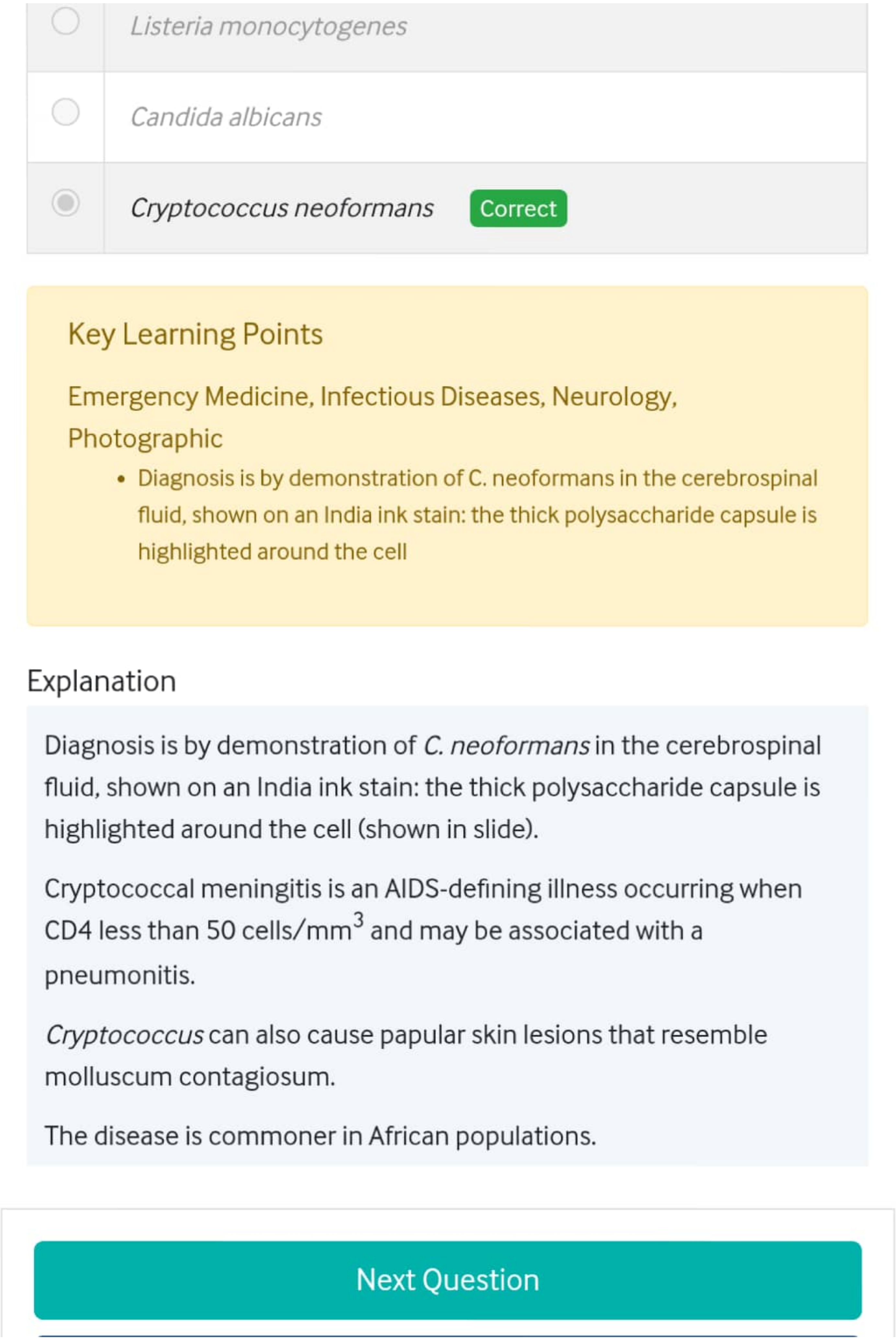
His wife said that he had been unwell for several weeks, but had complained of a high fever associated with neck stiffness for the past 24 hours. Microscopy of his CSF is pictured.



What organism is shown?

(Please select 1 option)

- ○ *Pneumocystis jirovecii*
- ○ *Toxoplasma gondii*
- ○ *Listeria monocytogenes*
- ○ *Candida albicans*
- ○ *Cryptococcus neoformans*



○ *Listeria monocytogenes*

○ *Candida albicans*

● *Cryptococcus neoformans* Correct

Key Learning Points

Emergency Medicine, Infectious Diseases, Neurology, Photographic

- Diagnosis is by demonstration of C. neoformans in the cerebrospinal fluid, shown on an India ink stain: the thick polysaccharide capsule is highlighted around the cell

Explanation

Diagnosis is by demonstration of *C. neoformans* in the cerebrospinal fluid, shown on an India ink stain: the thick polysaccharide capsule is highlighted around the cell (shown in slide).

Cryptococcal meningitis is an AIDS-defining illness occurring when CD4 less than 50 cells/mm^3 and may be associated with a pneumonitis.

Cryptococcus can also cause papular skin lesions that resemble molluscum contagiosum.

The disease is commoner in African populations.

A 19-year-old farm worker was admitted with a two day history of progressive weakness and tingling of the extremities affecting all limbs. He had been well until a week ago when he had an episode of running nose, cough and a sore throat which resolved after 48 hours without therapy.

On examination, pulse rate was 100 beats/min, BP 130/75 mmHg and respiratory rate 16/min. Cranial nerve examination was unremarkable. He had decreased muscle strength in all extremities, lower more than upper, and absent deep tendon reflexes in the legs.

Investigations showed:

Hb	146 g/L	(130-180)
WCC	9.3×10^9/L	(4-11)
Platelets	400×10^9/L	(150-400)
ESR	5 mm/hr	(0-15)
Plasma sodium	135 mmol/L	(137-144)
Plasma potassium	3.8 mmol/L	(3.5-4.9)
Plasma urea	6.0 mmol/L	(2.5-7.5)
Plasma creatinine	100 μmol/L	(60-110)
Plasma glucose	5.0 mmol/L	(3.0-6.0)
CSF opening pressure	150 mm H_2O	(50-180)
CSF cell count	<2 cells/ml	
CSF protein	1.5 g/l	(0.15-0.45)
CSF glucose	6.1 mmol/L	(3.3-4.4)

What is the most likely diagnosis?

(Please select 1 option)

- ○ Epidural spinal abscess
- ○ Post-infectious polyradiculopathy
- ○ Polymyositis
- ○ Central pontine myelinolysis
- ○ Viral encephalitis

Submit Answer

● Post-infectious polyradiculopathy **Correct**

○ Polymyositis

○ Central pontine myelinolysis

○ Viral encephalitis

Key Learning Points

Neurology

- A history of progressive weakness and loss of tendon reflexes associated with a history of recent infection suggests Guillain-Barré syndrome (post-infectious polyradiculopathy).

Explanation

The history of progressive weakness and loss of tendon reflexes associated with a history of recent infection suggests Guillain-Barré syndrome (post-infectious polyradiculopathy).

Respiratory function monitoring is crucial and best done by regularly measuring the vital capacity.

"The differential diagnosis of peripheral neuropathy is significantly narrowed by a focused clinical assessment that addresses several key issues"[1]

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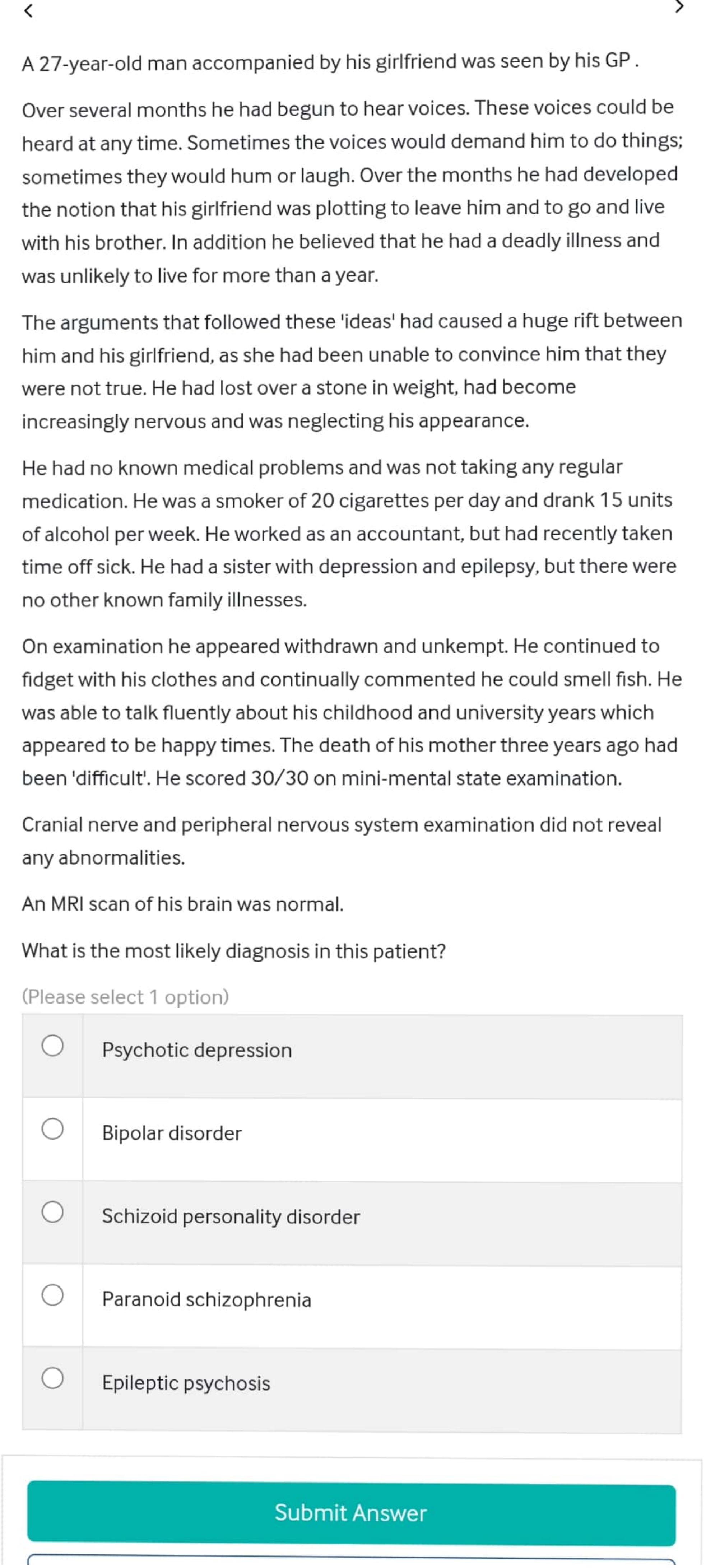

A 27-year-old man accompanied by his girlfriend was seen by his GP .

Over several months he had begun to hear voices. These voices could be heard at any time. Sometimes the voices would demand him to do things; sometimes they would hum or laugh. Over the months he had developed the notion that his girlfriend was plotting to leave him and to go and live with his brother. In addition he believed that he had a deadly illness and was unlikely to live for more than a year.

The arguments that followed these 'ideas' had caused a huge rift between him and his girlfriend, as she had been unable to convince him that they were not true. He had lost over a stone in weight, had become increasingly nervous and was neglecting his appearance.

He had no known medical problems and was not taking any regular medication. He was a smoker of 20 cigarettes per day and drank 15 units of alcohol per week. He worked as an accountant, but had recently taken time off sick. He had a sister with depression and epilepsy, but there were no other known family illnesses.

On examination he appeared withdrawn and unkempt. He continued to fidget with his clothes and continually commented he could smell fish. He was able to talk fluently about his childhood and university years which appeared to be happy times. The death of his mother three years ago had been 'difficult'. He scored 30/30 on mini-mental state examination.

Cranial nerve and peripheral nervous system examination did not reveal any abnormalities.

An MRI scan of his brain was normal.

What is the most likely diagnosis in this patient?

(Please select 1 option)

- ○ Psychotic depression
- ○ Bipolar disorder
- ○ Schizoid personality disorder
- ○ Paranoid schizophrenia
- ○ Epileptic psychosis

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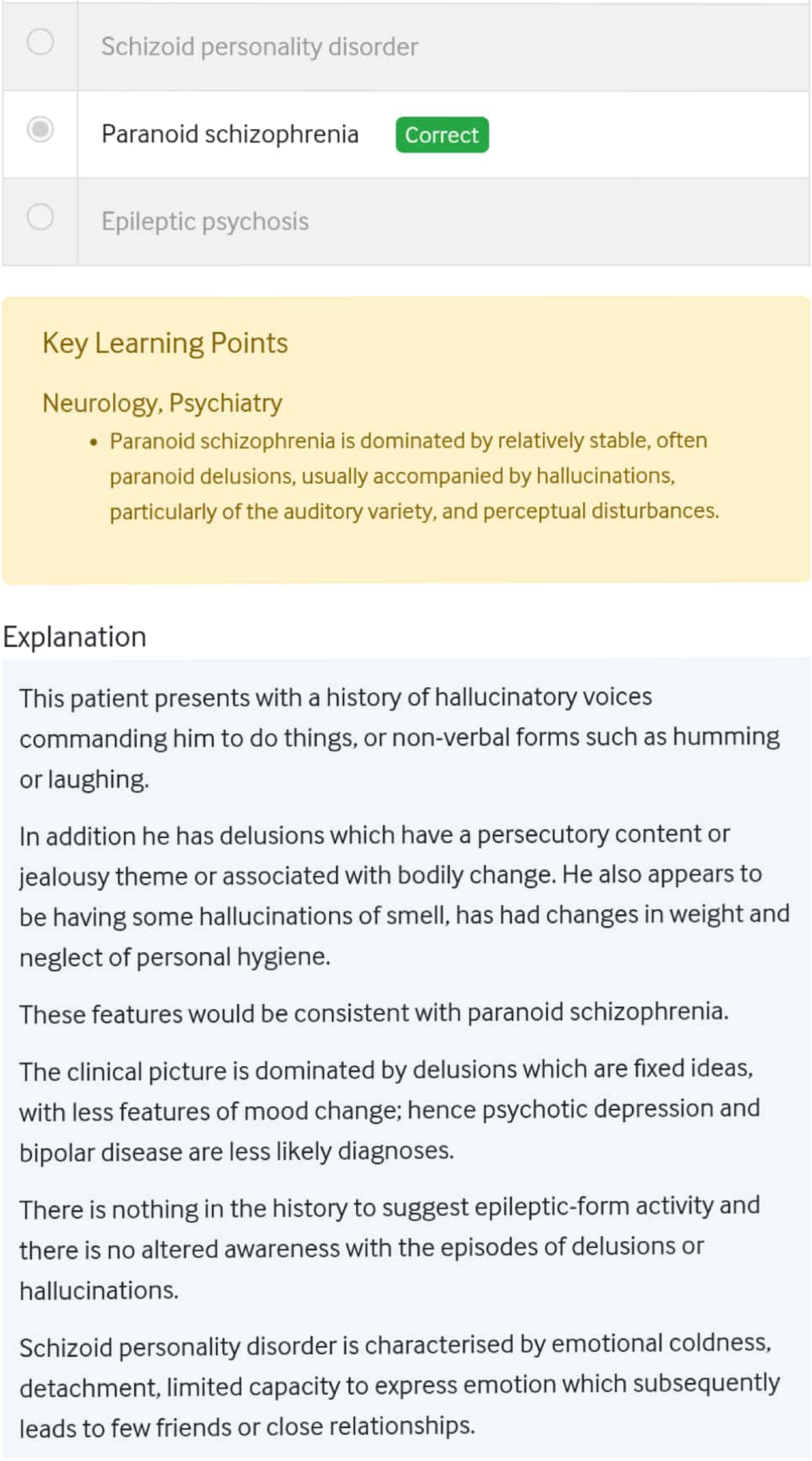

○ Schizoid personality disorder

● Paranoid schizophrenia **Correct**

○ Epileptic psychosis

Key Learning Points

Neurology, Psychiatry

- Paranoid schizophrenia is dominated by relatively stable, often paranoid delusions, usually accompanied by hallucinations, particularly of the auditory variety, and perceptual disturbances.

Explanation

This patient presents with a history of hallucinatory voices commanding him to do things, or non-verbal forms such as humming or laughing.

In addition he has delusions which have a persecutory content or jealousy theme or associated with bodily change. He also appears to be having some hallucinations of smell, has had changes in weight and neglect of personal hygiene.

These features would be consistent with paranoid schizophrenia.

The clinical picture is dominated by delusions which are fixed ideas, with less features of mood change; hence psychotic depression and bipolar disease are less likely diagnoses.

There is nothing in the history to suggest epileptic-form activity and there is no altered awareness with the episodes of delusions or hallucinations.

Schizoid personality disorder is characterised by emotional coldness, detachment, limited capacity to express emotion which subsequently leads to few friends or close relationships.

A 54-year-old woman was admitted to the Emergency department having developed progressive weakness and numbness affecting both legs.

Her symptoms appeared to have come on over a period of a week. Within the last few days she had noticed numbness and weakness affecting her hands and had also developed some double vision and weakness of facial muscles. She did not complain of any pain in her legs or hands. Prior to the development of her symptoms, she had been seen by her GP for shingles.

Her past medical history included rheumatoid arthritis and recently diagnosed diabetes secondary to long term steroids. Her medication was metformin 1.5 g/day and prednisolone 5 mg/day.

On examination she had cushingoid features. Her blood pressure was 145/85 mmHg (supine) and 120/65 mmHg (erect). Her pulse was 88/min and oxygen saturation was 98%. Chest expansion was 5 cm and breath count was 22. Examining cranial nerves, she had restricted left lateral gaze on testing ocular movements and bilateral facial weakness and dysarthria. Pupillary responses and fundoscopy were normal.

On examination of the upper limb, tone was reduced bilaterally and there was marked weakness of handgrip and wrist flexion/extension bilaterally. There was no fatigability. All reflexes were absent. In addition there was sensory loss to all modalities extending up to the elbow bilaterally. Examination of the lower limb revealed flaccid tone bilaterally with some wasting and weakness of the proximal muscles. There was marked weakness of dorsiflexion and plantarflexion of the foot bilaterally and no evidence of fatigability. All reflexes were absent, and there was reduced sensation to all sensory modalities extending to the knee bilaterally.

Blood tests showed:

Sodium	140 mmol/L	(137 - 144)
Potassium	3.4 mmol/L	(3.5 - 4.9)
Urea	5.6 mmol/L	(2.5 - 7.5)
Creatinine	111 µmol/L	(60 - 110)
HbA_{1c}	6.9%	(3.8 - 6.4)
	52 mmol/mol	(20 - 46)
Erythrocyte sedimentation rate	32mm/1st hour	(0 - 30)

Lumbar puncture showed:

Opening pressure	13 cmH_2O	(5 - 18)
CSF protein	1.30 g/L	(0.15 - 0.45)
CSF glucose	3.4 mmol/L	(3.3 - 4.4)
CSF white cell count	5 per ml	(≤5)
CSF red cell count	3 per ml	(≤5)

Given the above history and clinical findings, how would you initially manage this patient?

(Please select 1 option)

- ○ Arrange electromyography
- ○ Arrange nerve conduction studies
- ○ Commence on IV immunoglobulin at 0.4 g/Kg/day for five days
- ○ Commence on pyridostigmine 60 mg QDS
- ○ Commence on azathioprine 1 mg/kg/day

Submit Answer

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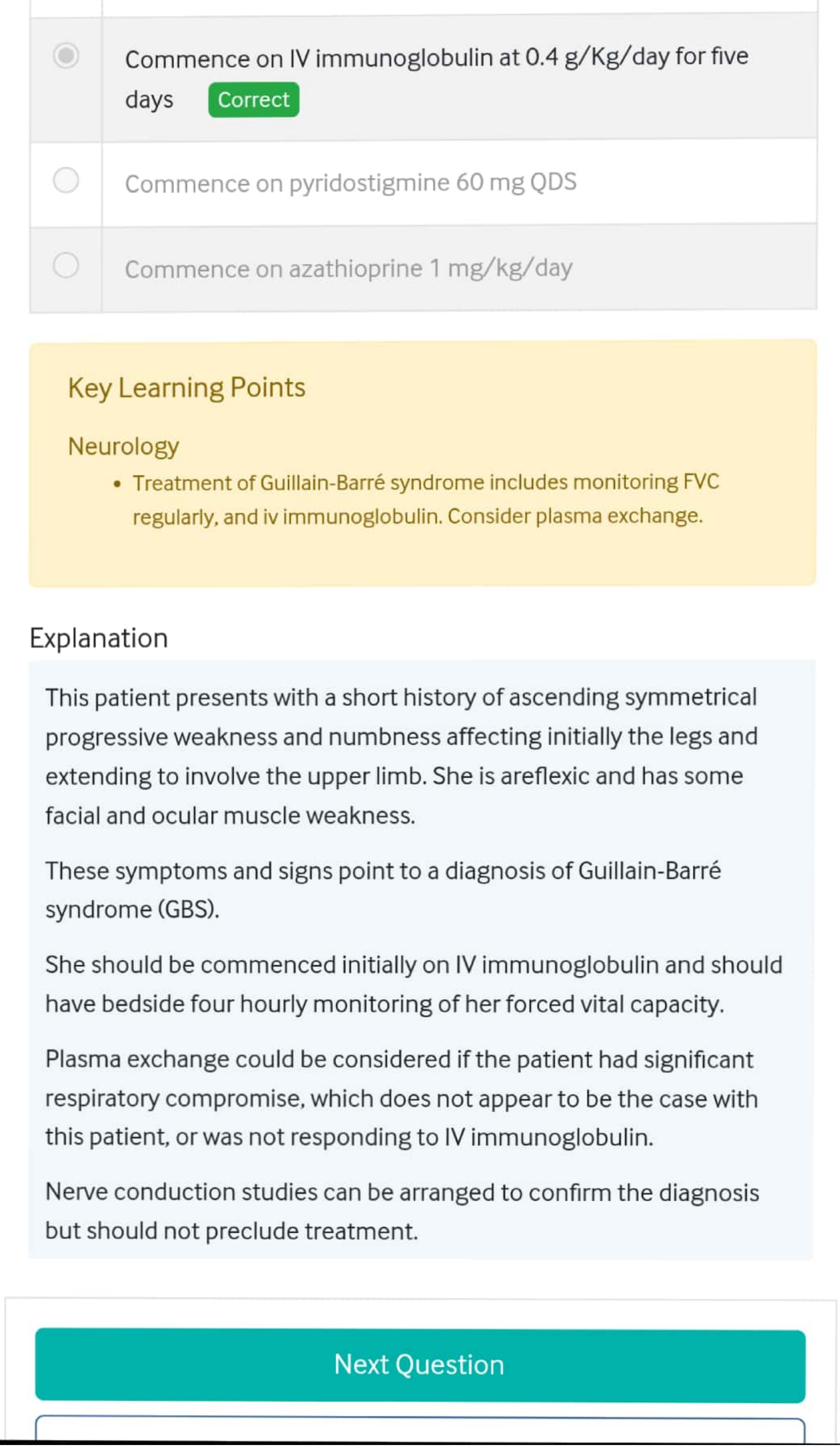


● Commence on IV immunoglobulin at 0.4 g/Kg/day for five days

○ Commence on pyridostigmine 60 mg QDS

○ Commence on azathioprine 1 mg/kg/day

Key Learning Points

Neurology

- Treatment of Guillain-Barré syndrome includes monitoring FVC regularly, and iv immunoglobulin. Consider plasma exchange.

Explanation

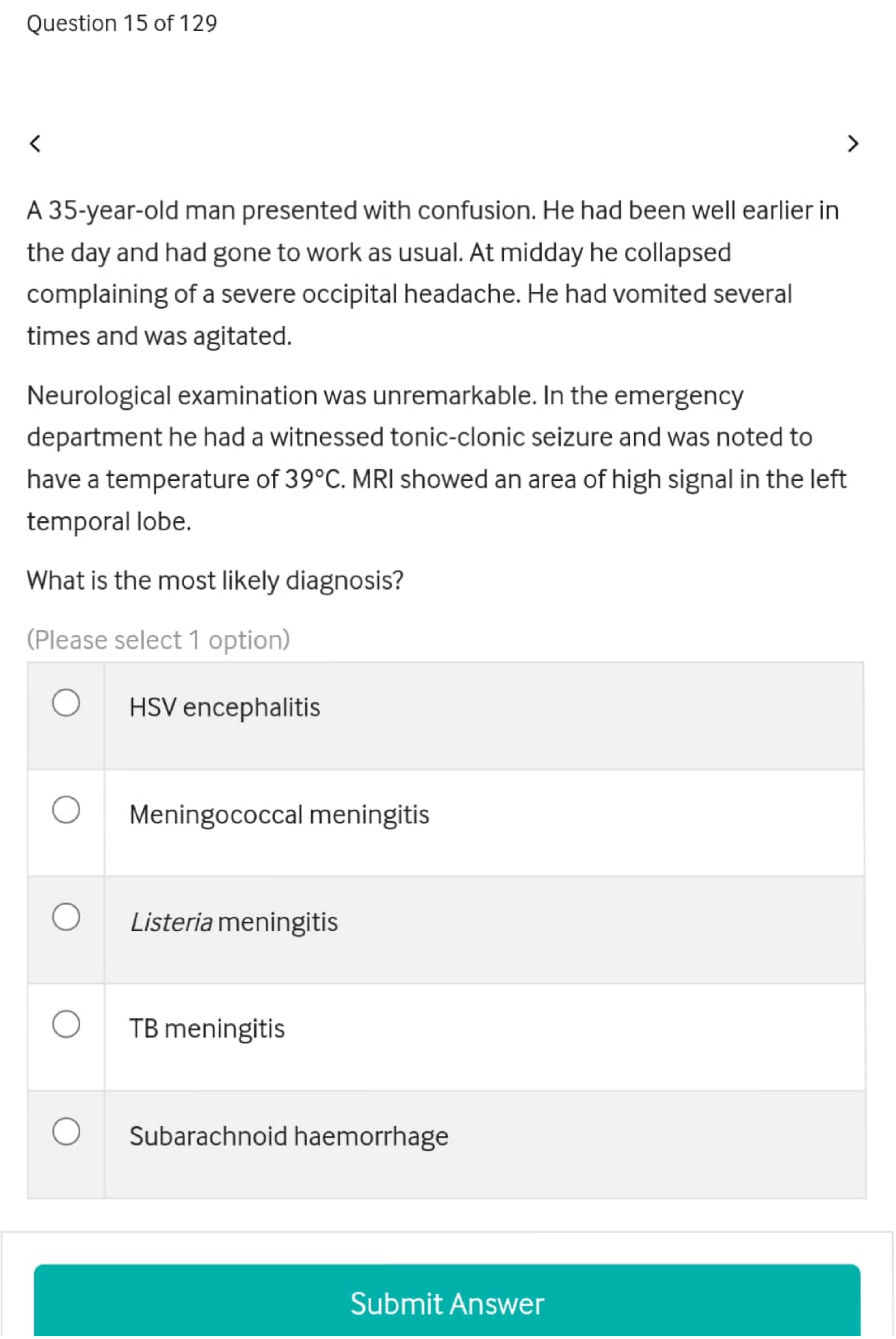
This patient presents with a short history of ascending symmetrical progressive weakness and numbness affecting initially the legs and extending to involve the upper limb. She is areflexic and has some facial and ocular muscle weakness.

These symptoms and signs point to a diagnosis of Guillain-Barré syndrome (GBS).

She should be commenced initially on IV immunoglobulin and should have bedside four hourly monitoring of her forced vital capacity.

Plasma exchange could be considered if the patient had significant respiratory compromise, which does not appear to be the case with this patient, or was not responding to IV immunoglobulin.

Nerve conduction studies can be arranged to confirm the diagnosis but should not preclude treatment.



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A 35-year-old man presented with confusion. He had been well earlier in the day and had gone to work as usual. At midday he collapsed complaining of a severe occipital headache. He had vomited several times and was agitated.

Neurological examination was unremarkable. In the emergency department he had a witnessed tonic-clonic seizure and was noted to have a temperature of 39°C. MRI showed an area of high signal in the left temporal lobe.

What is the most likely diagnosis?

(Please select 1 option)

○	HSV encephalitis
○	Meningococcal meningitis
○	*Listeria* meningitis
○	TB meningitis
○	Subarachnoid haemorrhage

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● HSV encephalitis **Correct**

○ Meningococcal meningitis

○ *Listeria* meningitis

○ TB meningitis

○ Subarachnoid haemorrhage

Key Learning Points

Neurology

- MRI findings in HSV encephalitis classically shows high signal in the temporal lobes.

Explanation

The most likely diagnosis is HSV encephalitis which presents with fever, headache, vomiting, behavioural changes, confusion and seizures. MRI classically shows high signal in the temporal lobes.

Listeria meningitis affects immunosuppressed patients or patients at extremes of age. It can present with reduced conscious level, seizures and cranial nerve palsies.

Meningococcal meningitis presents with headache, neck stiffness, photophobia, vomiting and fever.

Subarachnoid haemorrhage classically presents with a thunderclap headache and neck stiffness.

TB meningitis presents gradually with headache, neck stiffness, photophobia, vomiting and fever. It occurs more commonly in those who are immunosuppressed, for example secondary to AIDS.

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A 57-year-old man was seen by his GP complaining of weight loss and weakness. He had noticed this for some time and commented that his clothes do not fit him any more.

He had noticed progressive weakness affecting both his legs together with wasting of the thighs and thinning around the ankles. In addition he has noticed slurring of speech and whilst eating has had some nasal regurgitation. His past medical history included chronic bronchitis, prostatic hypertrophy and osteoarthritis affecting the cervical and lumbar spine. He was taking a salbutamol and beclasone inhaler.

On examination he was cachectic with muscle wasting of the arms and legs. Chest and abdominal examination appeared normal. On examination of the cranial nerves there was a pseudobulbar palsy, decreased palatal elevation and wasting of the tongue with fasciculations. In addition he had a brisk jaw jerk.

Examining the upper limb revealed normal tone, however there was asymmetrical wasting of the forearms and dorsal interossei bilaterally. There was asymmetrical weakness of hand grip (R > L) and also weakness of wrist flexion/extension. Reflexes all appeared pathologically brisk and there were positive Hoffman signs bilaterally.

Examining the lower limb revealed bilaterally increased tone with fasciculations of the thighs. There was marked weakness of dorsiflexion and plantar flexion of the left foot and bilateral weakness of hip flexion that was more marked on the left than the right. Sensory examination and cerebellar function was normal.

Blood tests:

Sodium	134 mmol/L	(137-144)
Potassium	3.7 mmol/L	(3.5-4.9)
Urea	5.7 mmol/L	(2.5-7.5)
Creatinine	98 µmol/L	(60-110)
Calcium	2.56 mmol/L	(2.2-2.6)
Liver function tests	Normal	-
Prostate specific antigen	3 g/L	(<4)

MR brain	Normal
MR spine	Lumbar and cervical degenerative disc disease affecting L3-S1 and C5-8
Chest x ray	Normal

Electromyography and nerve conduction studies show spontaneous fasciculation potentials affecting all four limbs.

Given the history and clinical findings so far, what is the likely cause for this patient's symptoms?

(Please select 1 option)

- ○ Paraneoplastic syndrome
- ○ Benign fasciculation syndrome
- ○ Eaton-Lambert syndrome
- ○ Motor neurone disease (MND)
- ○ Cervical myelopathy secondary to osteoarthritis and degenerative disc disease

Submit Answer

● Motor neurone disease (MND) **Correct**

○ Cervical myelopathy secondary to osteoarthritis and degenerative disc disease

Key Learning Points

Neurology

- MND will present with a mix of both upper and lower motor neuron signs without sensory disturbance.

Explanation

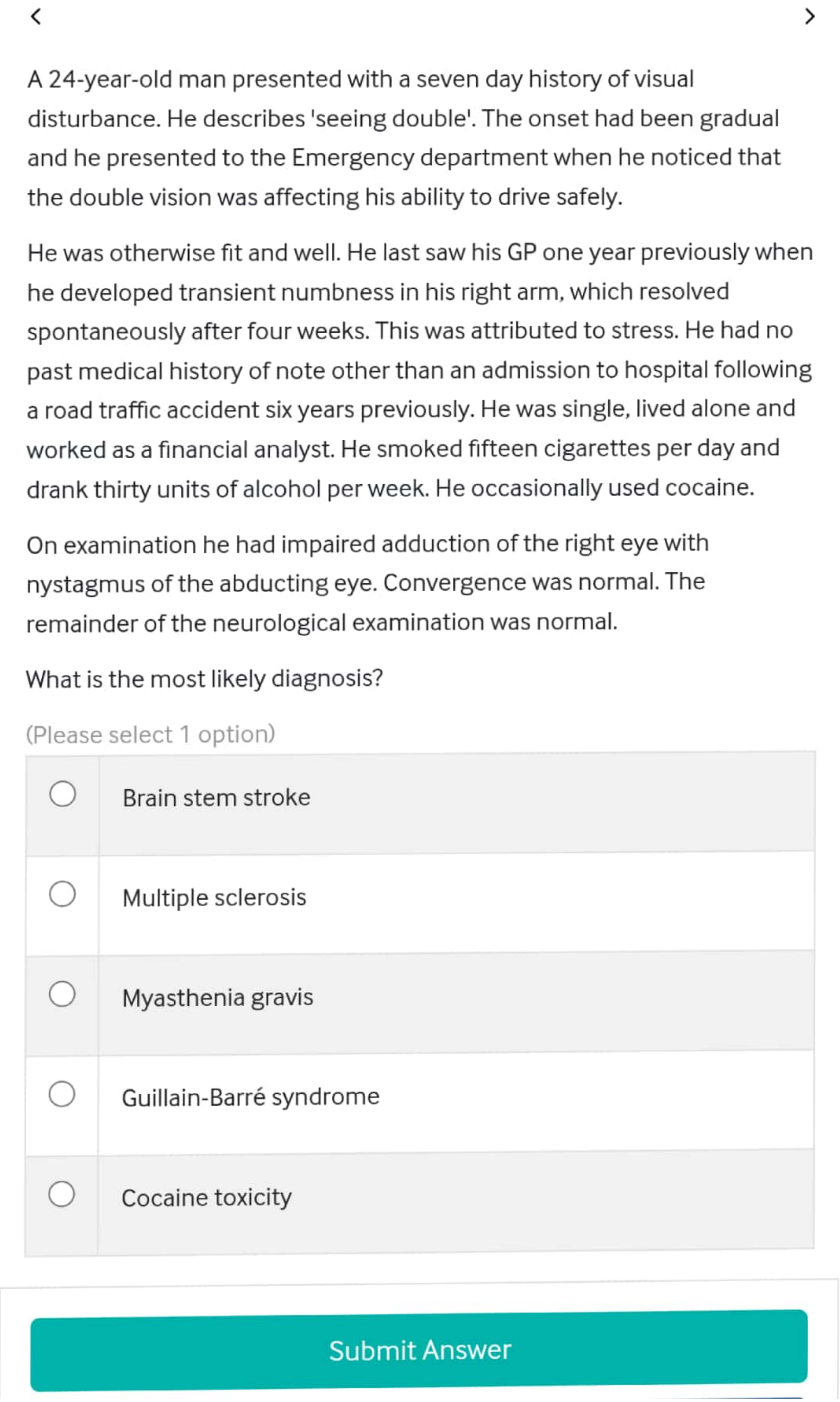
This patient has a history compatible with motor neurone disease (MND), presenting with a combination of upper and lower motor neurone signs, weight loss and generalised fasciculations. There is bulbar weakness and tongue wasting causing nasal regurgitation and dysarthria.

Cervical myelopathy can cause a combination of upper and lower motor neurone signs, with upper motor neurone signs in the legs and depending on the level of the lesion a combination of upper and lower motor neurone signs in the arms. It would not explain the bulbar weakness, tongue fasciculations and brisk jaw jerk however.

Benign fasciculation syndrome is another MND mimic disorder; however there is absence of hard neurological signs such as weakness, altered tone or reflexes.

Eaton-Lambert syndrome presents with a 'myasthenic' type clinical picture of fatigable weakness, together with depressed reflexes, which help differentiate it from myasthenia gravis. This is not the case with this patient.

A paraneoplastic disorder associated with an underlying malignancy can cause a whole host of neurological sequelae. Common paraneoplastic presentations include cerebellar syndrome and peripheral neuropathies. There is no suggestion in the history or clinical findings of an underlying malignancy.



A 24-year-old man presented with a seven day history of visual disturbance. He describes 'seeing double'. The onset had been gradual and he presented to the Emergency department when he noticed that the double vision was affecting his ability to drive safely.

He was otherwise fit and well. He last saw his GP one year previously when he developed transient numbness in his right arm, which resolved spontaneously after four weeks. This was attributed to stress. He had no past medical history of note other than an admission to hospital following a road traffic accident six years previously. He was single, lived alone and worked as a financial analyst. He smoked fifteen cigarettes per day and drank thirty units of alcohol per week. He occasionally used cocaine.

On examination he had impaired adduction of the right eye with nystagmus of the abducting eye. Convergence was normal. The remainder of the neurological examination was normal.

What is the most likely diagnosis?

(Please select 1 option)

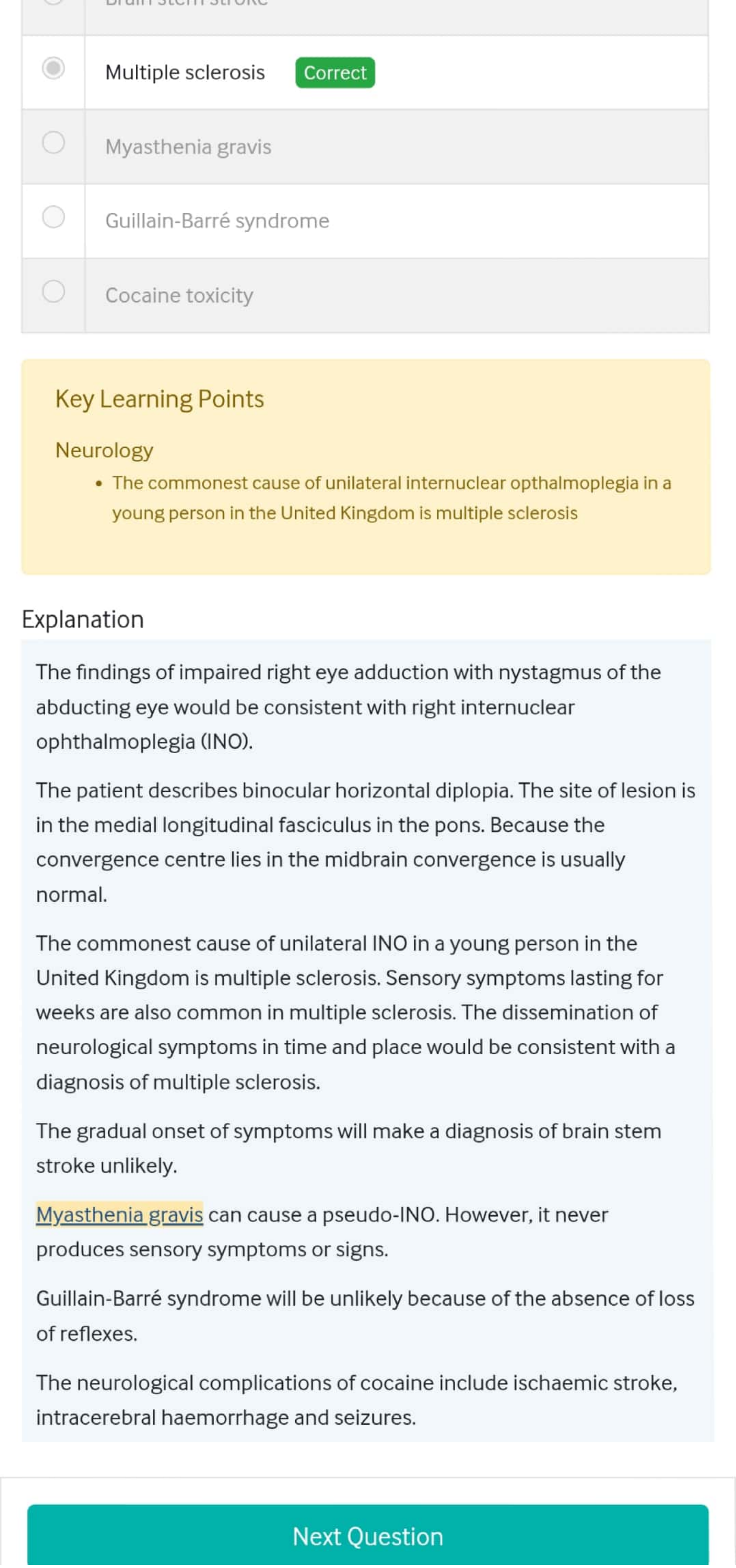
○ Brain stem stroke

○ Multiple sclerosis

○ Myasthenia gravis

○ Guillain-Barré syndrome

○ Cocaine toxicity



○ Brain stem stroke

● Multiple sclerosis **Correct**

○ Myasthenia gravis

○ Guillain-Barré syndrome

○ Cocaine toxicity

Key Learning Points

Neurology

- The commonest cause of unilateral internuclear opthalmoplegia in a young person in the United Kingdom is multiple sclerosis

Explanation

The findings of impaired right eye adduction with nystagmus of the abducting eye would be consistent with right internuclear ophthalmoplegia (INO).

The patient describes binocular horizontal diplopia. The site of lesion is in the medial longitudinal fasciculus in the pons. Because the convergence centre lies in the midbrain convergence is usually normal.

The commonest cause of unilateral INO in a young person in the United Kingdom is multiple sclerosis. Sensory symptoms lasting for weeks are also common in multiple sclerosis. The dissemination of neurological symptoms in time and place would be consistent with a diagnosis of multiple sclerosis.

The gradual onset of symptoms will make a diagnosis of brain stem stroke unlikely.

Myasthenia gravis can cause a pseudo-INO. However, it never produces sensory symptoms or signs.

Guillain-Barré syndrome will be unlikely because of the absence of loss of reflexes.

The neurological complications of cocaine include ischaemic stroke, intracerebral haemorrhage and seizures.

A late middle-aged homeless man is brought to the emergency department with a six hour history of profuse vomiting. He complains of nausea and headache.

The history available is sketchy. He is of no fixed abode and denies having any previous medical problems. He appears unkempt and is confused - oriented to person but not time or place. He is afebrile. His breath smells of ketones.

Twelve hours after admission his condition deteriorates. He complains of blurred vision and his pupils are fixed and dilated; his respiratory rate increases sharply over the next few minutes and he becomes unconscious.

Investigations show:

Hb	138 g/L	(130-180)
WCC	7.1 ×10^9/L	(4-11)
Platelets	401 ×10^9/L	(150-400)
Plasma sodium	135 mmol/L	(137-144)
Plasma potassium	5.0 mmol/L	(3.5-4.9)
Plasma urea	5.8 mmol/L	(2.5-7.5)
Plasma creatinine	110 µmol/L	(60-110)
Plasma chloride	100 mmol/L	(95-107)
Plasma bicarbonate	12 mmol/L	(20-28)
Plasma glucose	5.5 mmol/L	(3.0-6.0)
Plasma lactate	4.1 mmol/L	(0.6-1.7)
PaO_2	12 kPa	(11.3-12.6)
$PaCO_2$	4.2 kPa	(4.7-6.0)
pH	7.22	(7.36-7.44)
Urine microscopy	Crystals seen	

The anion gap is calculated as which of the following?

(Please select 1 option)

○ 28 mmol/L

○ 140 mmol/L

○ 112 mmol/L

○ 88 mmol/L

○ 12 mmol/L

Submit Answer

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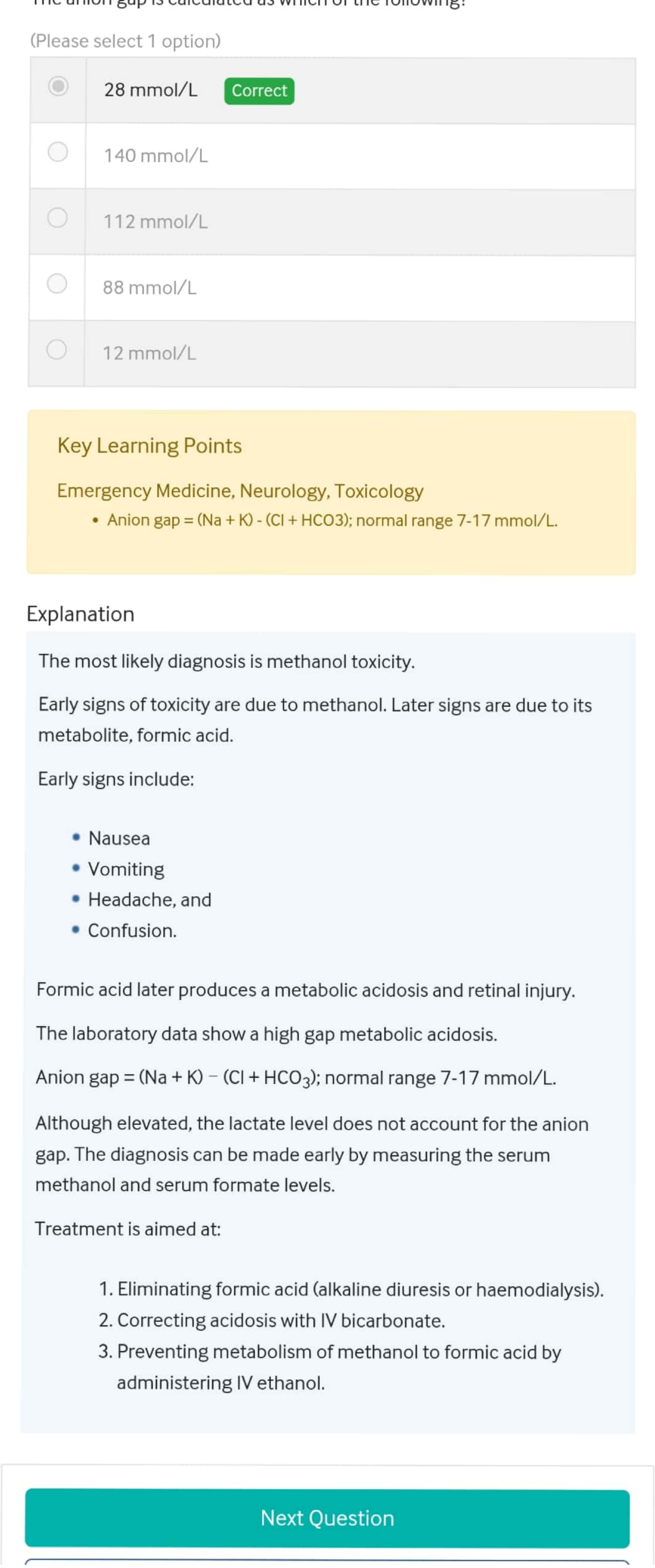


The anion gap is calculated as which of the following?

(Please select 1 option)

● 28 mmol/L **Correct**

○ 140 mmol/L

○ 112 mmol/L

○ 88 mmol/L

○ 12 mmol/L

Key Learning Points

Emergency Medicine, Neurology, Toxicology

- Anion gap = (Na + K) - (Cl + HCO3); normal range 7-17 mmol/L.

Explanation

The most likely diagnosis is methanol toxicity.

Early signs of toxicity are due to methanol. Later signs are due to its metabolite, formic acid.

Early signs include:

- Nausea
- Vomiting
- Headache, and
- Confusion.

Formic acid later produces a metabolic acidosis and retinal injury.

The laboratory data show a high gap metabolic acidosis.

Anion gap = (Na + K) − (Cl + HCO_3); normal range 7-17 mmol/L.

Although elevated, the lactate level does not account for the anion gap. The diagnosis can be made early by measuring the serum methanol and serum formate levels.

Treatment is aimed at:

1. Eliminating formic acid (alkaline diuresis or haemodialysis).
2. Correcting acidosis with IV bicarbonate.
3. Preventing metabolism of methanol to formic acid by administering IV ethanol.

Question 19 of 129

‹ ›

A 14-year-old girl presents with a seven day history of unsteadiness, diplopia, dizziness and drowsiness. Prior to that she has been well and has achieved average performances at school.

She takes no medication and her last menstrual period was two weeks ago. Family history reveals that her mother has hypothyroidism and her father has a history of epilepsy.

On examination, she seems well but there is coarse nystagmus, dysarthria and a wide gait ataxia.

Investigations show a normal full blood count, renal function and liver function tests. Chest x ray is normal. Both CT and MRI brain are normal. Lumbar puncture shows normal pressure, cells, protein and glucose.

What is the most likely diagnosis?

(Please select 1 option)

○ Meningitis

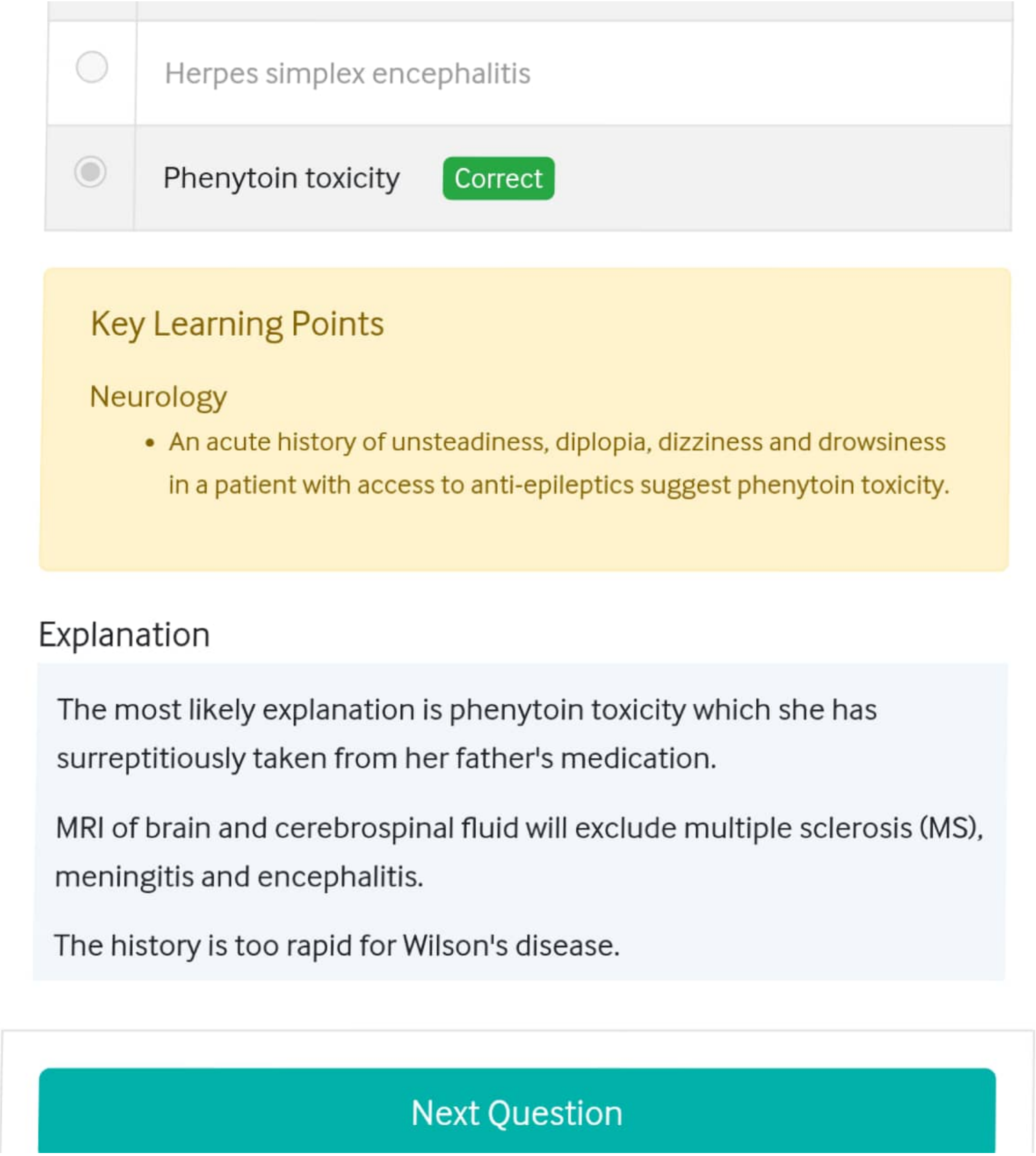
○ Wilson’s disease

○ Multiple sclerosis

○ Herpes simplex encephalitis

○ Phenytoin toxicity

Submit Answer



○ Herpes simplex encephalitis

● Phenytoin toxicity

Key Learning Points

Neurology

- An acute history of unsteadiness, diplopia, dizziness and drowsiness in a patient with access to anti-epileptics suggest phenytoin toxicity.

Explanation

The most likely explanation is phenytoin toxicity which she has surreptitiously taken from her father's medication.

MRI of brain and cerebrospinal fluid will exclude multiple sclerosis (MS), meningitis and encephalitis.

The history is too rapid for Wilson's disease.

Question 20

Question 20 of 129

<   >

A 35-year-old man with a history of schizophrenia presented following an overdose of his own medications. He became progressively confused, ataxic, and febrile.

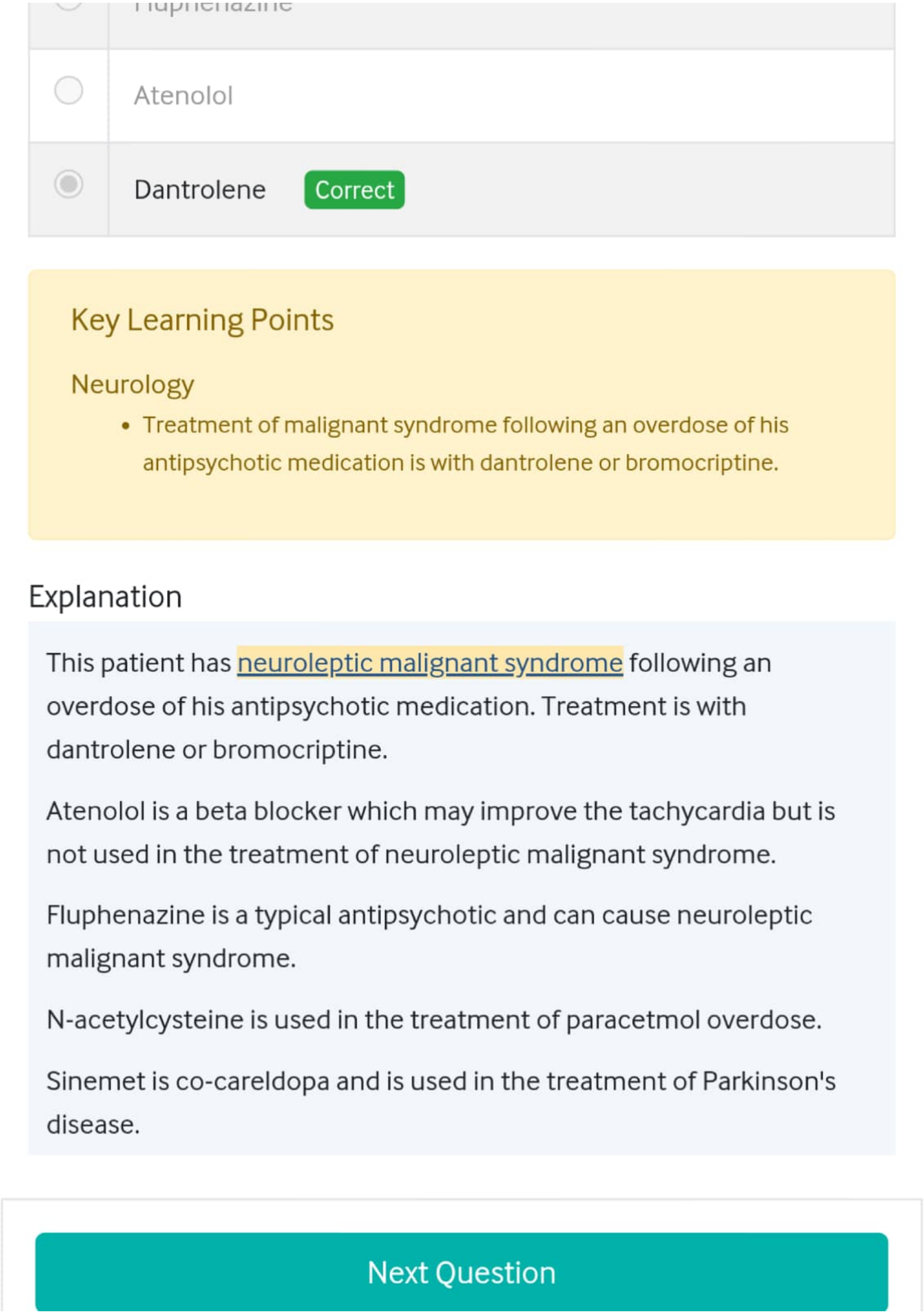
On examination there was increased tone in his limbs with lead-pipe rigidity. ECG revealed a sinus tachycardia. Head CT, lumbar puncture and septic screen were negative.

Which drug may help this patient's condition?

(Please select 1 option)

○	N-acetylcysteine
○	Sinemet
○	Fluphenazine
○	Atenolol
○	Dantrolene

Submit Answer



○ Fluphenazine

○ Atenolol

● Dantrolene **Correct**

Key Learning Points

Neurology

- Treatment of malignant syndrome following an overdose of his antipsychotic medication is with dantrolene or bromocriptine.

Explanation

This patient has neuroleptic malignant syndrome following an overdose of his antipsychotic medication. Treatment is with dantrolene or bromocriptine.

Atenolol is a beta blocker which may improve the tachycardia but is not used in the treatment of neuroleptic malignant syndrome.

Fluphenazine is a typical antipsychotic and can cause neuroleptic malignant syndrome.

N-acetylcysteine is used in the treatment of paracetmol overdose.

Sinemet is co-careldopa and is used in the treatment of Parkinson's disease.

A 63-year-old man was admitted to hospital with headache, nausea, confusion, somnolence and paresis of his left leg. The weakness had been progressively getting worse over several weeks; however the confusion and headache had come on over the last five days. He was being investigated for weight loss and shortness of breath.

He had a past medical history of arthritis in the knees and hands. Apart from taking occasional paracetamol he did not take any other regularly prescribed medications. He was a non-smoker and did not drink alcohol. He had been looking after his mother who had recently died of breast carcinoma. He had worked as an electrician all his life.

On examination he appeared confused and disorientated. His blood pressure was 158/89 mmHg and pulse was 62 beats per minute and regular and temperature was 36.8°C. Heart sounds appeared normal. There was evidence of axillary and cervical lymphadenopathy and a mass over the left chest. Auscultation of the chest revealed some scattered crackles but no other features. Abdominal examination was normal. Neurological examination revealed some neck stiffness. Fundoscopy and the rest of the cranial nerve examination were normal. Examination of the peripheral nervous system revealed a left-sided lower limb paresis with hypertonia and hyperreflexia. There were no demonstratable sensory or cerebellar findings.

He underwent a number of investigations which are outlined below. A lumbar puncture was contraindicated.

Sodium	134 mmol/L	(137-144)
Potassium	3.3 mmol/L	(3.5-4.9)
Urea	8.9 mmol/L	(2.5-7.5)
Creatinine	120 μmol/L	(60-110)
Calcium	3.2 mmol/L	(2.2-2.6)
Glucose	5.4 mmol/L	(3.0-6.0)
Angiotensin-converting enzyme	80 U/L	(25-82)
Haemoglobin	116 g/L	(130-180)
White cell count	7.8 $\times 10^9$/L	(4-11)
Platelets	156 $\times 10^9$/L	(150-400)
Mean cell volume	82 fL	(80-96)
C reactive protein	10 mg/L	(<10)
Antineutrophil cytoplasmic antibodies	Negative	

A chest x ray showed multiple lung lesions.

An MRI scan of the brain showed meningeal enhancement, enhancement of the basilar cisterns and multiple enhancing masses with marked surrounding oedema.

What is the most likely diagnosis?

(Please select 1 option)

○	Neurosarcoidosis
○	Tuberculous meningitis
○	Brain metastases and lepto-meningeal carcinomatosis
○	Granulomatosis with polyangiitis
○	Bacterial meningitis

Submit Answer

○ Tuberculous meningitis

○ Brain metastases and lepto-meningeal carcinomatosis
This is the correct answer

○ Granulomatosis with polyangiitis

○ Bacterial meningitis

Explanation

The most likely diagnosis that would fit all symptoms, signs and investigations would be brain metastases and lepto-meningeal carcinomatosis. There is a suggestion that this may be emanating from a left breast carcinoma. There are also lung metastases and lymphadenopathy.

Bacterial meningitis is unlikely given the prolonged history, focal neurology and normal white cell count and inflammatory markers.

Neurosarcoidosis tends to present with mononeuropathies, cranial nerve palsies and cerebral or cerebellar dysfunction and rarely meningitis. Angiotensin-converting enzyme (ACE) levels would be typically elevated and lung features would include bilateral hilar lymphadenopathy or interstitial changes.

There is nothing in the history to suggest a risk of tuberculosis.

Granulomatosis with polyangiitis typically affects the ears, nose, throat and upper respiratory airways with neurological sequelae manifesting as mononeuropathies.

Male breast cancer is a rare disease that is diagnosed mainly in elderly men (median age 63-67 years). Risk factors might include positive family history (mother or sister), less physical activity and higher weight and body mass index than average.

Next Question

A 35-year-old man was admitted to hospital having developed sudden onset quadriparesis, associated with complete loss of speech.

His partner who had been present at the time stated that he had been well and had not complained of any symptoms prior to the event. He had no past medical history and did not take any prescribed medications. He was however a frequent user of cocaine and occasionally injected heroin.

On admission he was found to have a Glasgow coma scale of 3/15 (V1, M1, E1). There was no nuchal rigidity or rash. His blood pressure was 138/89 mmHg, pulse was 78 beats per minute and regular temperature was 36.8°C and blood glucose monitoring read a level of 5.6 mmol/L.

General, cardiovascular and respiratory examinations were normal. Neurological examination revealed pinpoint pupils with oculoparesis of horizontal movements on oculocephalic testing. Limb examination revealed generalised hypertonia and hyperreflexia with bilateral extensor plantar responses. He was intubated and taken to intensive care.

Over the next four weeks he was extensively investigated. He subsequently regained consciousness although was unable to speak and exhibited decerebrate rigidity. He could open his eyes and look up and down, but was unable to make any other purposeful response.

What is the most likely aetiology for this patient's condition?

(Please select 1 option)

○ Supratentorial mass with secondary brainstem compression

○ Opiate overdose

○ Venous sinus thrombosis

○ Pontine haemorrhage

○ Meningoencephalitis

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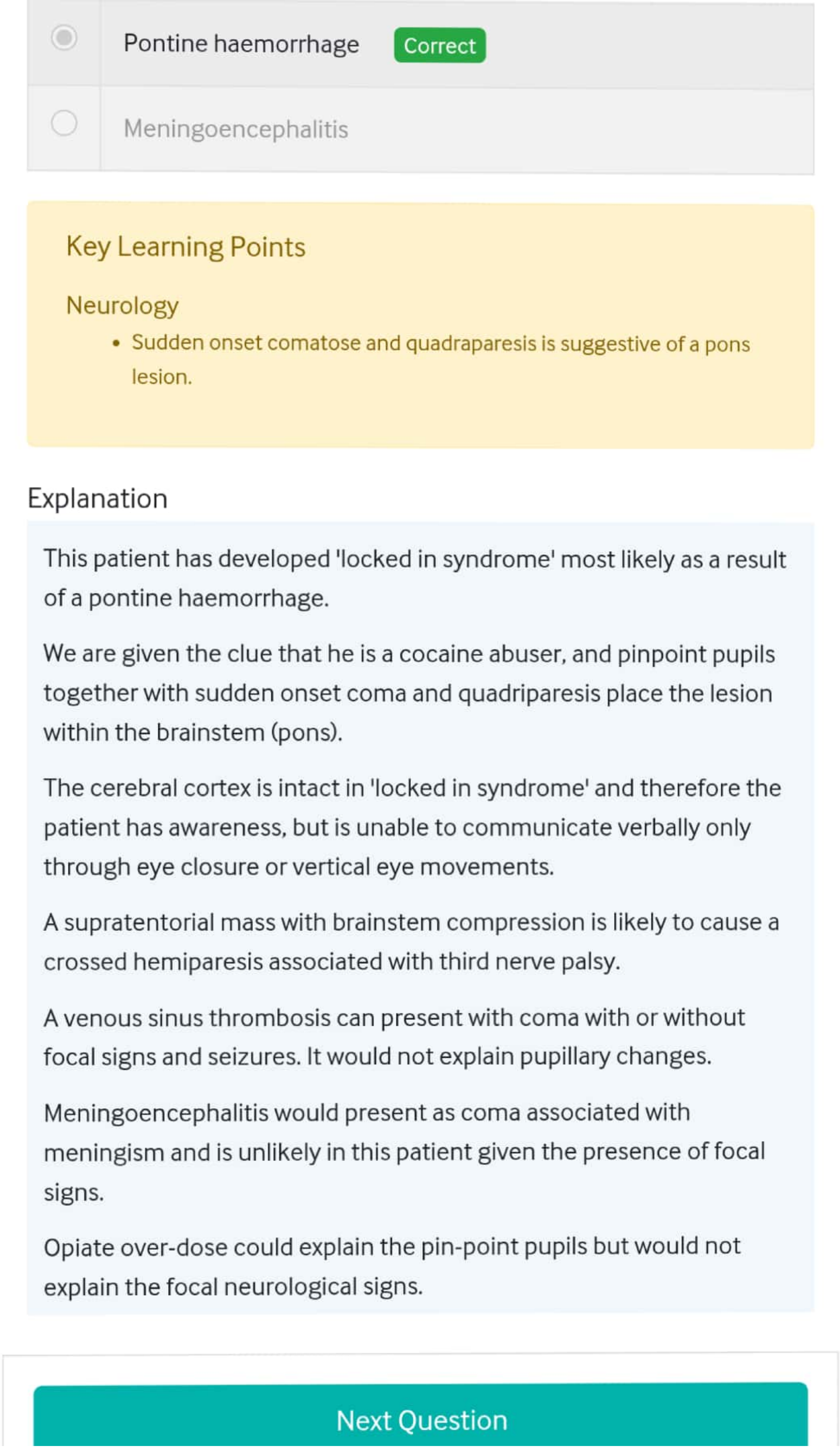


● Pontine haemorrhage **Correct**

○ Meningoencephalitis

Key Learning Points

Neurology

- Sudden onset comatose and quadraparesis is suggestive of a pons lesion.

Explanation

This patient has developed 'locked in syndrome' most likely as a result of a pontine haemorrhage.

We are given the clue that he is a cocaine abuser, and pinpoint pupils together with sudden onset coma and quadriparesis place the lesion within the brainstem (pons).

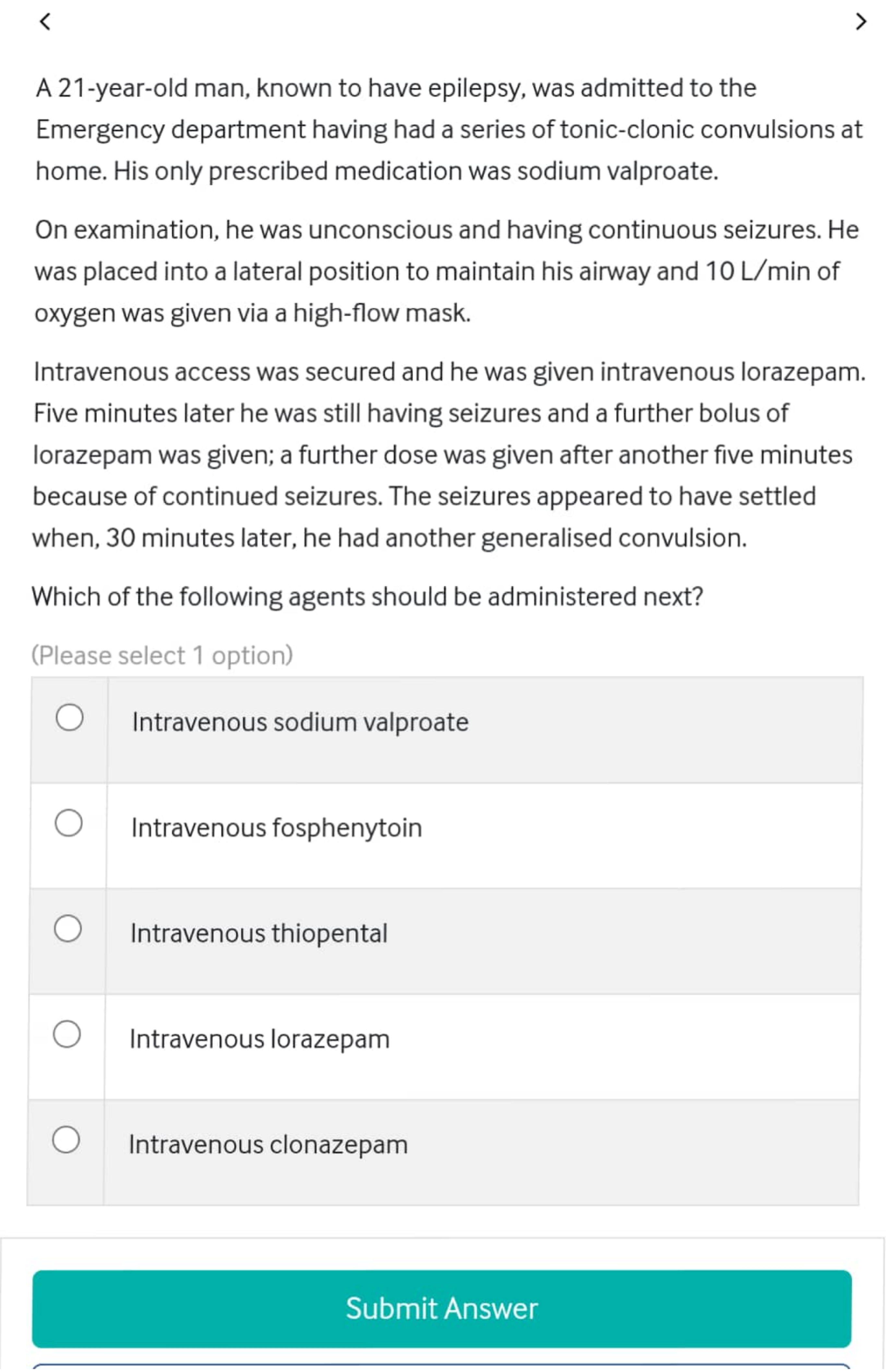
The cerebral cortex is intact in 'locked in syndrome' and therefore the patient has awareness, but is unable to communicate verbally only through eye closure or vertical eye movements.

A supratentorial mass with brainstem compression is likely to cause a crossed hemiparesis associated with third nerve palsy.

A venous sinus thrombosis can present with coma with or without focal signs and seizures. It would not explain pupillary changes.

Meningoencephalitis would present as coma associated with meningism and is unlikely in this patient given the presence of focal signs.

Opiate over-dose could explain the pin-point pupils but would not explain the focal neurological signs.



A 21-year-old man, known to have epilepsy, was admitted to the Emergency department having had a series of tonic-clonic convulsions at home. His only prescribed medication was sodium valproate.

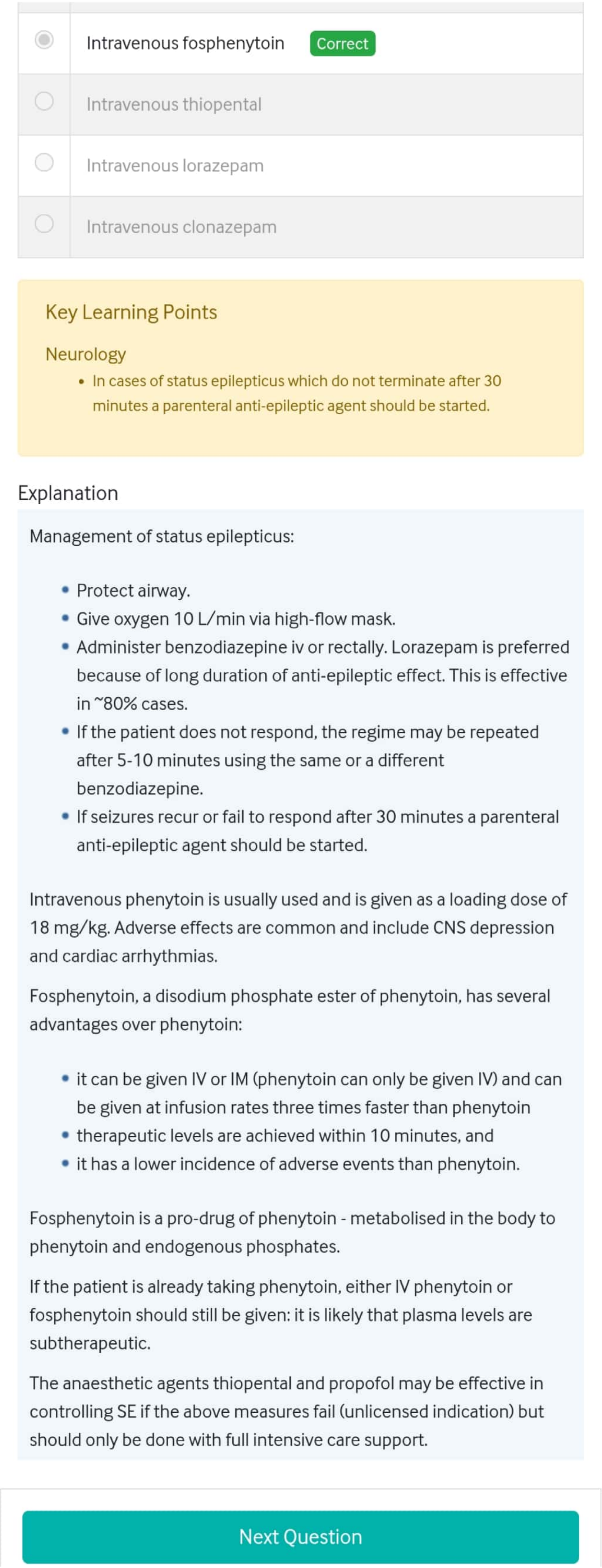
On examination, he was unconscious and having continuous seizures. He was placed into a lateral position to maintain his airway and 10 L/min of oxygen was given via a high-flow mask.

Intravenous access was secured and he was given intravenous lorazepam. Five minutes later he was still having seizures and a further bolus of lorazepam was given; a further dose was given after another five minutes because of continued seizures. The seizures appeared to have settled when, 30 minutes later, he had another generalised convulsion.

Which of the following agents should be administered next?

(Please select 1 option)

- ○ Intravenous sodium valproate
- ○ Intravenous fosphenytoin
- ○ Intravenous thiopental
- ○ Intravenous lorazepam
- ○ Intravenous clonazepam



● Intravenous fosphenytoin Correct

○ Intravenous thiopental

○ Intravenous lorazepam

○ Intravenous clonazepam

Key Learning Points

Neurology

- In cases of status epilepticus which do not terminate after 30 minutes a parenteral anti-epileptic agent should be started.

Explanation

Management of status epilepticus:

- Protect airway.
- Give oxygen 10 L/min via high-flow mask.
- Administer benzodiazepine iv or rectally. Lorazepam is preferred because of long duration of anti-epileptic effect. This is effective in ~80% cases.
- If the patient does not respond, the regime may be repeated after 5-10 minutes using the same or a different benzodiazepine.
- If seizures recur or fail to respond after 30 minutes a parenteral anti-epileptic agent should be started.

Intravenous phenytoin is usually used and is given as a loading dose of 18 mg/kg. Adverse effects are common and include CNS depression and cardiac arrhythmias.

Fosphenytoin, a disodium phosphate ester of phenytoin, has several advantages over phenytoin:

- it can be given IV or IM (phenytoin can only be given IV) and can be given at infusion rates three times faster than phenytoin
- therapeutic levels are achieved within 10 minutes, and
- it has a lower incidence of adverse events than phenytoin.

Fosphenytoin is a pro-drug of phenytoin - metabolised in the body to phenytoin and endogenous phosphates.

If the patient is already taking phenytoin, either IV phenytoin or fosphenytoin should still be given: it is likely that plasma levels are subtherapeutic.

The anaesthetic agents thiopental and propofol may be effective in controlling SE if the above measures fail (unlicensed indication) but should only be done with full intensive care support.

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A 25-year-old woman presented to her general practitioner with an eight week history of left leg weakness.

She had noticed that she had started to drag the leg while walking and had become increasingly unsteady on her feet. Two years previously she had presented to her GP complaining of patchy visual loss and orbital pain in the right eye. She had a past medical history of irritable bowel disease and tension headaches for which she was taking nadolol. She was a non-smoker and drank 8 units of alcohol per week. There was no family history of medical disorders.

On examination she was alert and orientated. Her blood pressure was 135/85 mmHg, temperature was 36.7°C, pulse 88 beats per minute and regular and heart sounds were normal. Cranial nerve examination appeared normal.

On examination of the upper limb, tone, power, reflexes and sensation were all normal. On lower limb examination tone was increased and there was 4/5 weakness of hip flexion, knee flexion and dorsiflexion. The knee and ankle jerk were both brisk and there was a left extensor plantar response. Sensation appeared intact. She appeared to be dragging her left leg on examining gait. Chest and abdominal examination was normal.

Investigations revealed:

Haemoglobin	123 g/L	(150-165)
White cell count	10.5×10^9/L	(4-11)
Platelets	355×10^9/L	(140-400)
Serum sodium	136 mmol/L	(137-144)
Serum potassium	4.5 mmol/L	(3.5-4.9)
Serum urea	4.6 mmol/L	(2.5-7.5)
Serum creatinine	88 µmol/L	(60-110)
Serum corrected calcium	2.5 mmol/L	(2.2-2.6)
Plasma glucose	4.6 mmol/L	(3.0-6.0)
Plasma cholesterol	3.6 mmol/L	(<5.2)
Erythrocyte sedimentation rate	5 mm in 1st hour	(0-20)
Serum angiotensin converting enzyme	35 U/L	(25-82)

A lumbar puncture was performed and the following results obtained:

Opening pressure	12 cmH_2O	(50-180)
CSF protein	0.5 g/L	(0.15-0.45)
CSF white cell count	5 cells per ml	(≤5)
CSF red cell count	4 cells per ml	(≤5)
CSF oligoclonal bands	Positive	-
CSF lactate	2.4 mmol/L	(1-2)
CSF angiotensin-converting enzyme	56 U/L	(20-94)
Serum oligoclonal bands	Negative	-

A T2-weighted MRI scan of the brain and spine showed multiple non-enhancing periventricular hyperintensities and gadolinium-enhancing lesions within the pons and cervical spine.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Multiple sclerosis
- ○ Vasculitis
- ○ Neurosarcoidosis
- ○ Acute disseminated encephalomyelitis
- ○ Anterior cerebral artery stroke

Submit Answer

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● Multiple sclerosis **Correct**

○ Vasculitis

○ Neurosarcoidosis

○ Acute disseminated encephalomyelitis

○ Anterior cerebral artery stroke

Key Learning Points

Neurology

- MS investigations will show intrathecal production of oligoclonal bands and multiple white matter lesions on T2-weighted MRI scan some of which appear gadolinium enhancing.

Explanation

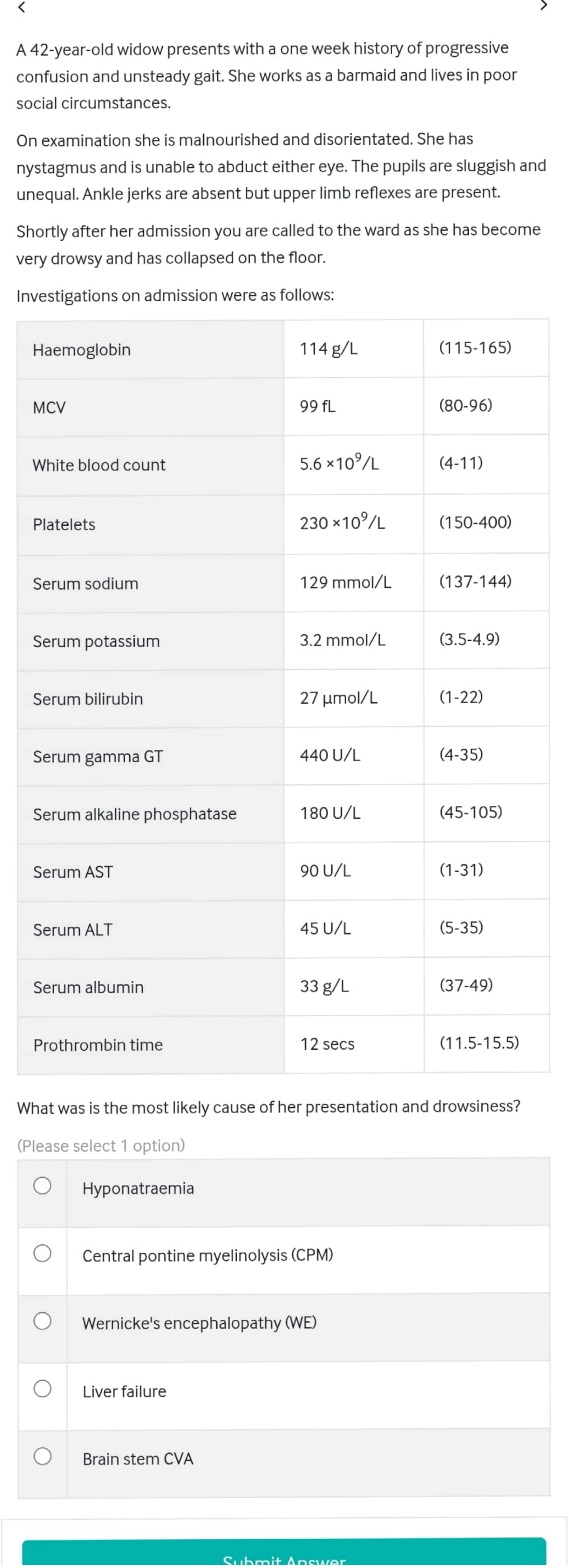
This patient presents with pyramidal weakness affecting the left leg and a prior history of right optic neuritis.

She has intrathecal production of oligoclonal bands and multiple white matter lesions on T2-weighted MRI scan some of which appear gadolinium enhancing. These findings would be in keeping with multiple sclerosis.

There is no history of infection or vaccination or altered conscious level or fever to suggest acute disseminated encephalomyelitis (ADEM) and not all lesions are enhancing.

She has no risk factors for cerebral stroke and no associated features for sarcoidosis.

Although she has a past history of headaches, her erythrocyte sedimentation rate is normal making vasculitis a less likely diagnosis.



A 42-year-old widow presents with a one week history of progressive confusion and unsteady gait. She works as a barmaid and lives in poor social circumstances.

On examination she is malnourished and disorientated. She has nystagmus and is unable to abduct either eye. The pupils are sluggish and unequal. Ankle jerks are absent but upper limb reflexes are present.

Shortly after her admission you are called to the ward as she has become very drowsy and has collapsed on the floor.

Investigations on admission were as follows:

Haemoglobin	114 g/L	(115-165)
MCV	99 fL	(80-96)
White blood count	5.6 ×10^9/L	(4-11)
Platelets	230 ×10^9/L	(150-400)
Serum sodium	129 mmol/L	(137-144)
Serum potassium	3.2 mmol/L	(3.5-4.9)
Serum bilirubin	27 µmol/L	(1-22)
Serum gamma GT	440 U/L	(4-35)
Serum alkaline phosphatase	180 U/L	(45-105)
Serum AST	90 U/L	(1-31)
Serum ALT	45 U/L	(5-35)
Serum albumin	33 g/L	(37-49)
Prothrombin time	12 secs	(11.5-15.5)

What was is the most likely cause of her presentation and drowsiness?

(Please select 1 option)

○ Hyponatraemia

○ Central pontine myelinolysis (CPM)

○ Wernicke's encephalopathy (WE)

○ Liver failure

○ Brain stem CVA

● Wernicke's encephalopathy (WE) Correct

○ Liver failure

○ Brain stem CVA

Key Learning Points

Neurology

- Encephalopathy, gait ataxia and oculomotor dysfunction with a background of alcohol misuse is suggestive of Wernicke's encephalopathy.

Explanation

This lady presents with the classic triad of WE:

- Encephalopathy
- Gait ataxia, and
- Oculomotor dysfunction.

Lower limb neuropathy is also a feature of WE.

Her occupation, poor nutrition, social situation, and blood results all suggest underlying alcoholism.

The hyponatraemia is mild and unlikely to cause symptoms and CPM (related to the rapid correction of Na+) is unlikely.

A brainstem cerebrovascular accident is unlikely due to the gradual onset over two weeks.

Liver function appears well preserved (normal prothrombin time and reasonable albumin).

A 60-year-old man was referred to clinic complaining of a constant headache and unsteadiness of gait. The unsteadiness had been progressive over a period of several weeks and his wife had noticed his speech had become slurred. More recently he had felt nauseated and had had several episodes of vomiting. He denied any weight loss, but had developed a dry cough.

He had a past medical history of hypertension treated with bendroflumethiazide 2.5 mg/day and epilepsy diagnosed at the age of 9 years for which he was taking phenytoin 300 mg/day. He was a heavy smoker of 40 cigarettes per day and drank 12 units of alcohol per week.

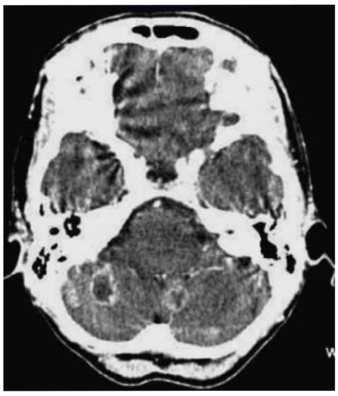
On examination he had a marked dysarthria. His blood pressure was 145/85 mmHg and pulse was 82 beats per minute and regular. Fundoscopy revealed bilateral papilloedema. He had reduced visual acuity of 6/9 in both eyes. The rest of the cranial nerves were normal and there was no evidence of neck stiffness.

On examination of the upper and lower limb, tone, power and reflexes all appeared normal; however he exhibited a marked intention tremor bilaterally and walked with a broad based ataxic gait. Chest examination revealed reduced air entry at the left base with a monophonic unilateral wheeze.

Investigations showed:

Haemoglobin	158 g/L	(130-180)
White cell count	13.4 $\times 10^9$/L	(4-11)
Platelets	425 $\times 10^9$/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	8.3 mmol/L	(2.5-7.5)
Serum creatinine	138 µmol/L	(60-110)
Serum corrected calcium	2.95 mmol/L	(2.2-2.6)
Plasma glucose	4.5 mmol/L	(3.0-6.0)
C reactive protein	10 mg/L	(<10)

A CT scan of his brain was performed. An image from this scan, following intravenous contrast, is shown below:



What is the likely diagnosis in this patient?

(Please select 1 option)

○ Metastases

○ Cerebellar infarct

○ Cerebellar abscess

○ Cerebellar astrocytoma

○ Phenytoin toxicity and benign haemangiomas

Submit Answer

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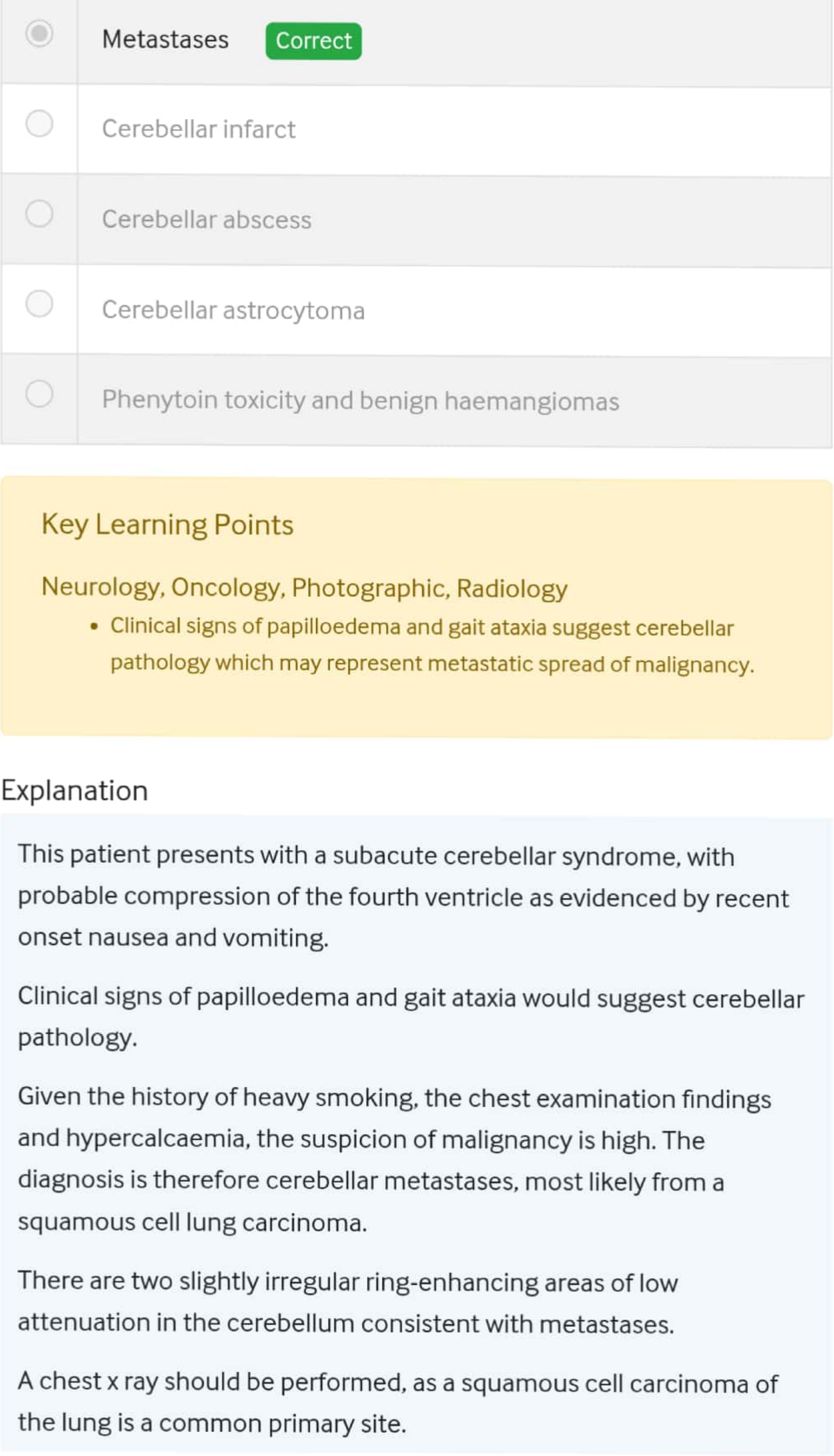

● Metastases **Correct**

○ Cerebellar infarct

○ Cerebellar abscess

○ Cerebellar astrocytoma

○ Phenytoin toxicity and benign haemangiomas

Key Learning Points

Neurology, Oncology, Photographic, Radiology

- Clinical signs of papilloedema and gait ataxia suggest cerebellar pathology which may represent metastatic spread of malignancy.

Explanation

This patient presents with a subacute cerebellar syndrome, with probable compression of the fourth ventricle as evidenced by recent onset nausea and vomiting.

Clinical signs of papilloedema and gait ataxia would suggest cerebellar pathology.

Given the history of heavy smoking, the chest examination findings and hypercalcaemia, the suspicion of malignancy is high. The diagnosis is therefore cerebellar metastases, most likely from a squamous cell lung carcinoma.

There are two slightly irregular ring-enhancing areas of low attenuation in the cerebellum consistent with metastases.

A chest x ray should be performed, as a squamous cell carcinoma of the lung is a common primary site.

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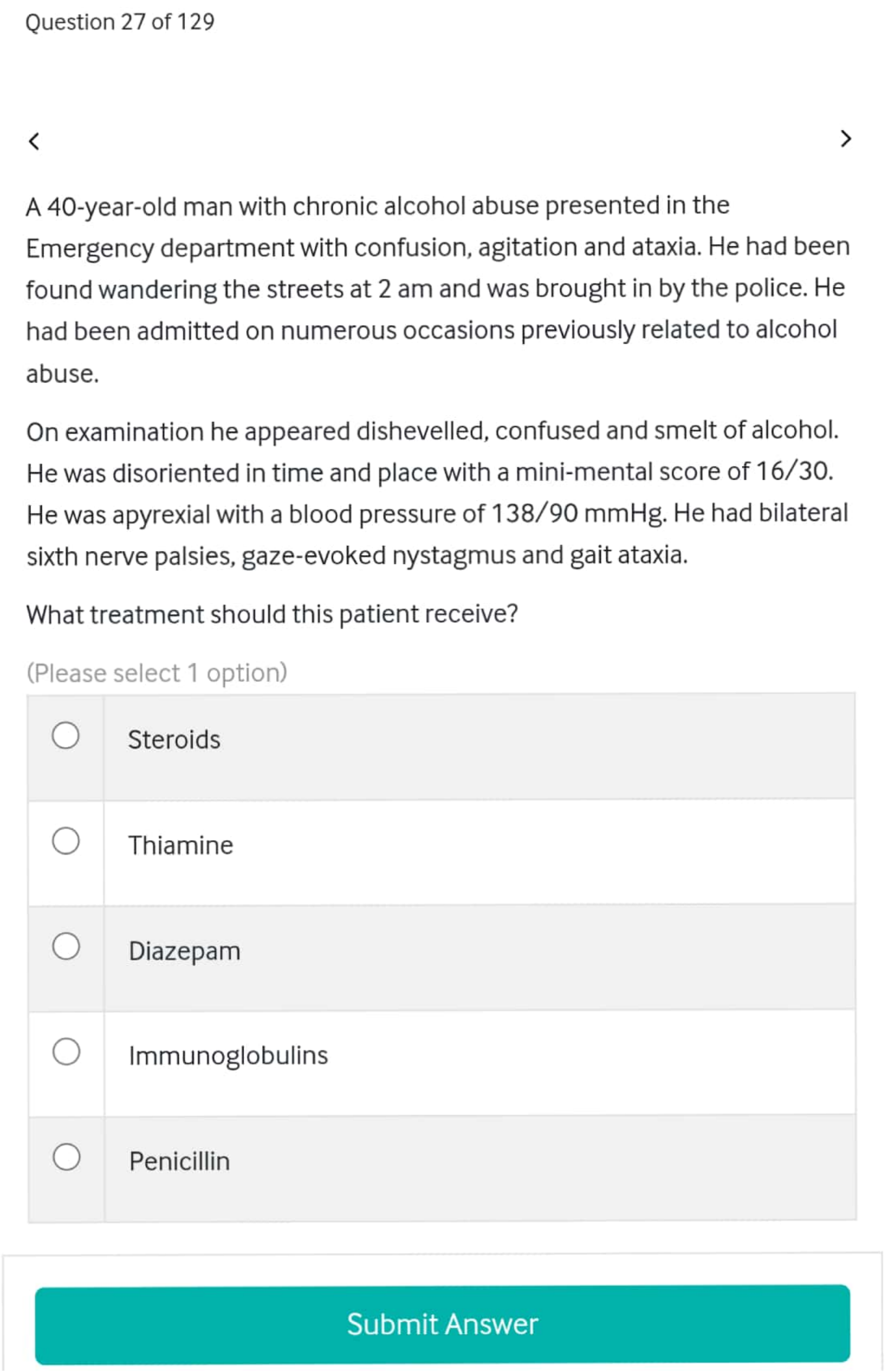

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A 40-year-old man with chronic alcohol abuse presented in the Emergency department with confusion, agitation and ataxia. He had been found wandering the streets at 2 am and was brought in by the police. He had been admitted on numerous occasions previously related to alcohol abuse.

On examination he appeared dishevelled, confused and smelt of alcohol. He was disoriented in time and place with a mini-mental score of 16/30. He was apyrexial with a blood pressure of 138/90 mmHg. He had bilateral sixth nerve palsies, gaze-evoked nystagmus and gait ataxia.

What treatment should this patient receive?

(Please select 1 option)

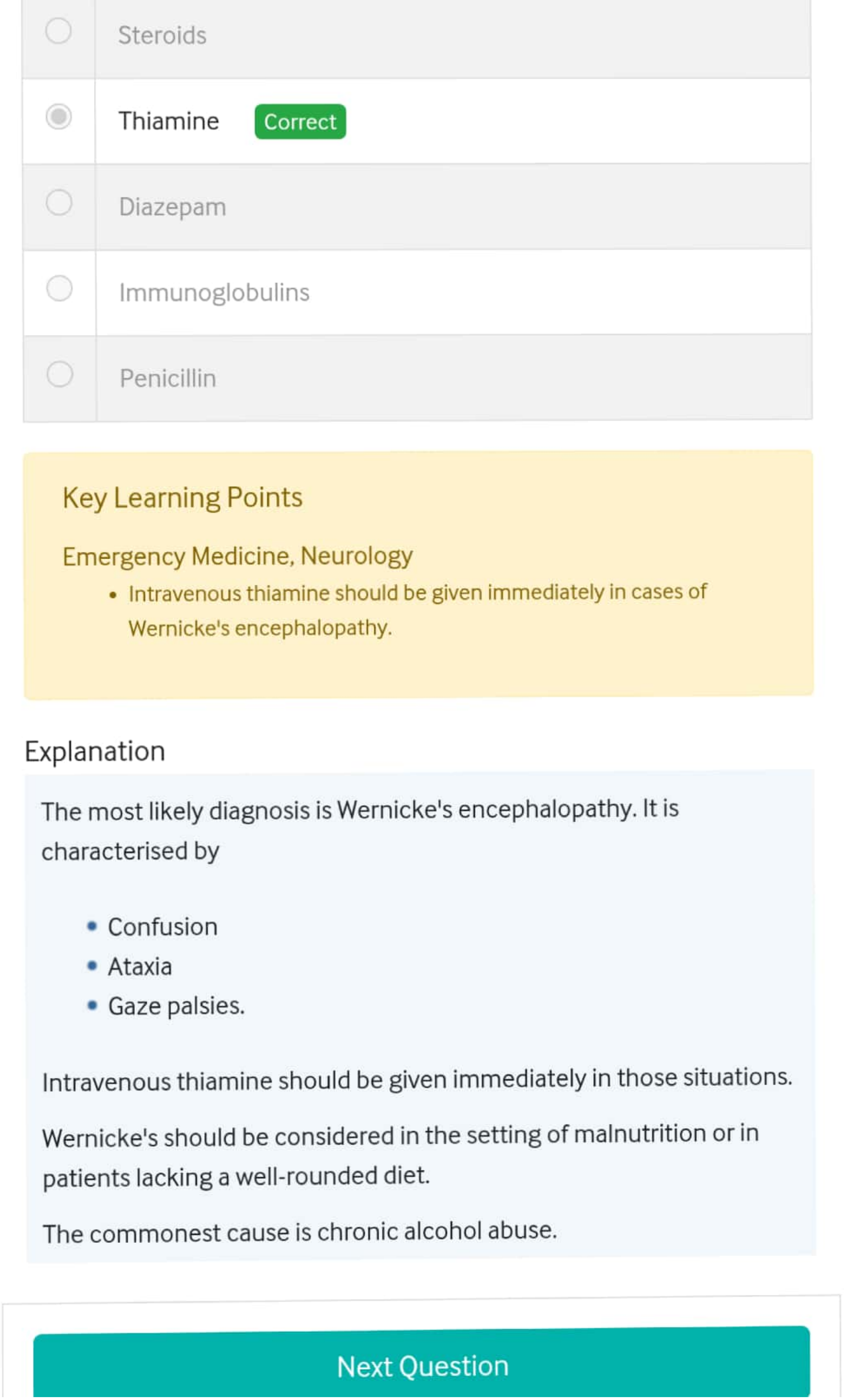
○ Steroids

○ Thiamine

○ Diazepam

○ Immunoglobulins

○ Penicillin



○ Steroids

● Thiamine Correct

○ Diazepam

○ Immunoglobulins

○ Penicillin

Key Learning Points

Emergency Medicine, Neurology

- Intravenous thiamine should be given immediately in cases of Wernicke's encephalopathy.

Explanation

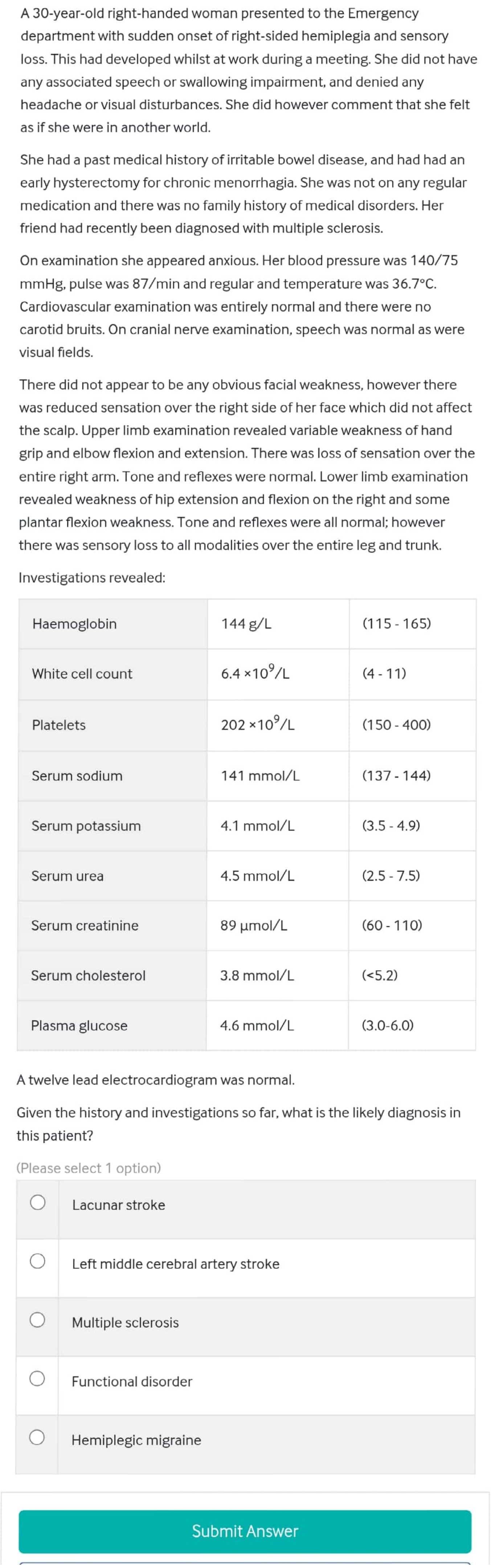
The most likely diagnosis is Wernicke's encephalopathy. It is characterised by

- Confusion
- Ataxia
- Gaze palsies.

Intravenous thiamine should be given immediately in those situations.

Wernicke's should be considered in the setting of malnutrition or in patients lacking a well-rounded diet.

The commonest cause is chronic alcohol abuse.



A 30-year-old right-handed woman presented to the Emergency department with sudden onset of right-sided hemiplegia and sensory loss. This had developed whilst at work during a meeting. She did not have any associated speech or swallowing impairment, and denied any headache or visual disturbances. She did however comment that she felt as if she were in another world.

She had a past medical history of irritable bowel disease, and had had an early hysterectomy for chronic menorrhagia. She was not on any regular medication and there was no family history of medical disorders. Her friend had recently been diagnosed with multiple sclerosis.

On examination she appeared anxious. Her blood pressure was 140/75 mmHg, pulse was 87/min and regular and temperature was 36.7°C. Cardiovascular examination was entirely normal and there were no carotid bruits. On cranial nerve examination, speech was normal as were visual fields.

There did not appear to be any obvious facial weakness, however there was reduced sensation over the right side of her face which did not affect the scalp. Upper limb examination revealed variable weakness of hand grip and elbow flexion and extension. There was loss of sensation over the entire right arm. Tone and reflexes were normal. Lower limb examination revealed weakness of hip extension and flexion on the right and some plantar flexion weakness. Tone and reflexes were all normal; however there was sensory loss to all modalities over the entire leg and trunk.

Investigations revealed:

Haemoglobin	144 g/L	(115 - 165)
White cell count	6.4 ×10^9/L	(4 - 11)
Platelets	202 ×10^9/L	(150 - 400)
Serum sodium	141 mmol/L	(137 - 144)
Serum potassium	4.1 mmol/L	(3.5 - 4.9)
Serum urea	4.5 mmol/L	(2.5 - 7.5)
Serum creatinine	89 µmol/L	(60 - 110)
Serum cholesterol	3.8 mmol/L	(<5.2)
Plasma glucose	4.6 mmol/L	(3.0-6.0)

A twelve lead electrocardiogram was normal.

Given the history and investigations so far, what is the likely diagnosis in this patient?

(Please select 1 option)

○ Lacunar stroke

○ Left middle cerebral artery stroke

○ Multiple sclerosis

○ Functional disorder

○ Hemiplegic migraine

○ Multiple sclerosis

● Functional disorder **Correct**

○ Hemiplegic migraine

Key Learning Points

Neurology

- Derealisation at the time of a suspect seizure is likely a symptom of a functional disorder.

Explanation

This patient presents with sudden onset right-sided weakness affecting the arm and leg in absence of the face, and a hemisensory disturbance.

She also gives a possible history of derealisation at the time of the event.

She has no cerebrovascular risk factors or any symptoms suggestive of migraine.

Her examination does not appear to relate to any particular pattern of weakness and she has an unusual distribution of sensory loss affecting the trunk in addition to the arm, leg and face excluding the scalp.

The most likely diagnosis is a functional disorder, although MRI brain imaging is necessary as thalamic strokes can rarely present with hemibody loss of sensation.

Exploration and investigation into psychological disturbances such as depression and anxiety are required in this patient.

Next Question

A 60-year-old man was brought to the Emergency department after a fall in his bathroom.

Seen immediately by his family, he was already picking himself up from the floor and said he was not injured. His wife felt that he was transiently dazed.

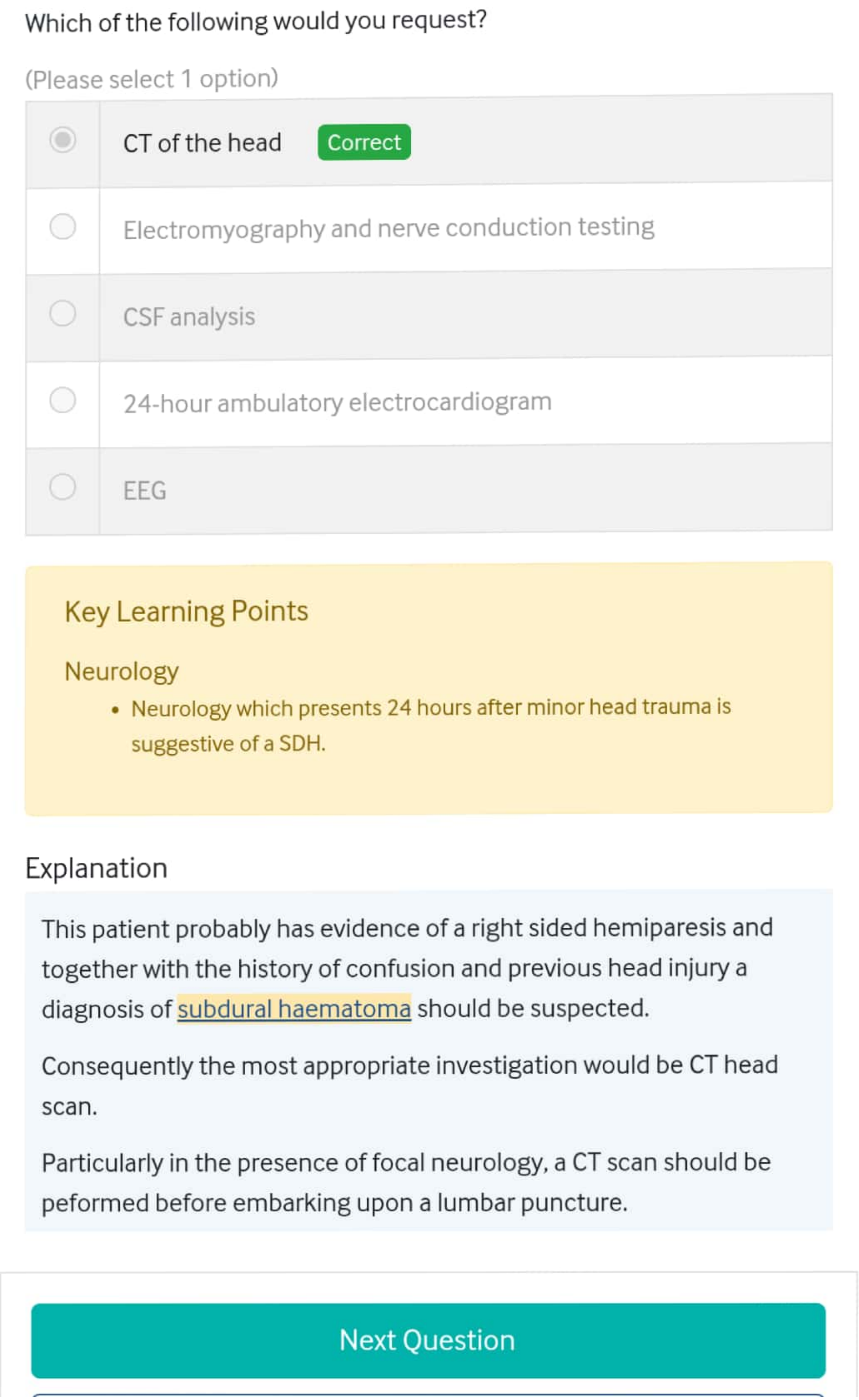
On examination, he was alert, and no abnormalities were noted. His medical history included a history of hypertension for which he was taking bendroflumethiazide 2.5 mg daily. He was discharged without any further intervention.

Two weeks later his wife brings the patient to see you because the dazed state has returned. Examination reveals a temperature of 36.7°C, a pulse rate of 84 bpm regular, a blood pressure of 152/94 mm Hg. On questioning he is slightly slowed, being disoriented to time with some deficit in recent memory. The patient moves slowly, but power is normal. Neurologic examination shows slight hyperactivity of the tendon reflexes on the right with unclear plantar responses because of bilateral withdrawal.

Which of the following would you request?

(Please select 1 option)

○	CT of the head
○	Electromyography and nerve conduction testing
○	CSF analysis
○	24-hour ambulatory electrocardiogram
○	EEG



Which of the following would you request?

(Please select 1 option)

- ● CT of the head **Correct**
- ○ Electromyography and nerve conduction testing
- ○ CSF analysis
- ○ 24-hour ambulatory electrocardiogram
- ○ EEG

Key Learning Points

Neurology

- Neurology which presents 24 hours after minor head trauma is suggestive of a SDH.

Explanation

This patient probably has evidence of a right sided hemiparesis and together with the history of confusion and previous head injury a diagnosis of subdural haematoma should be suspected.

Consequently the most appropriate investigation would be CT head scan.

Particularly in the presence of focal neurology, a CT scan should be peformed before embarking upon a lumbar puncture.

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A 26-year-old male epileptic is admitted with temperature and rash.

Over the last one week a rash has developed and he has become increasingly ill. Recently he has had some problems with epileptic control and has commenced carbamazepine with valproate.

Examination reveals an unwell patient with a temperature of 39°C, a diffuse erythematous, painful rash with evidence of some lateral sliding of these erythematous areas on palpation.

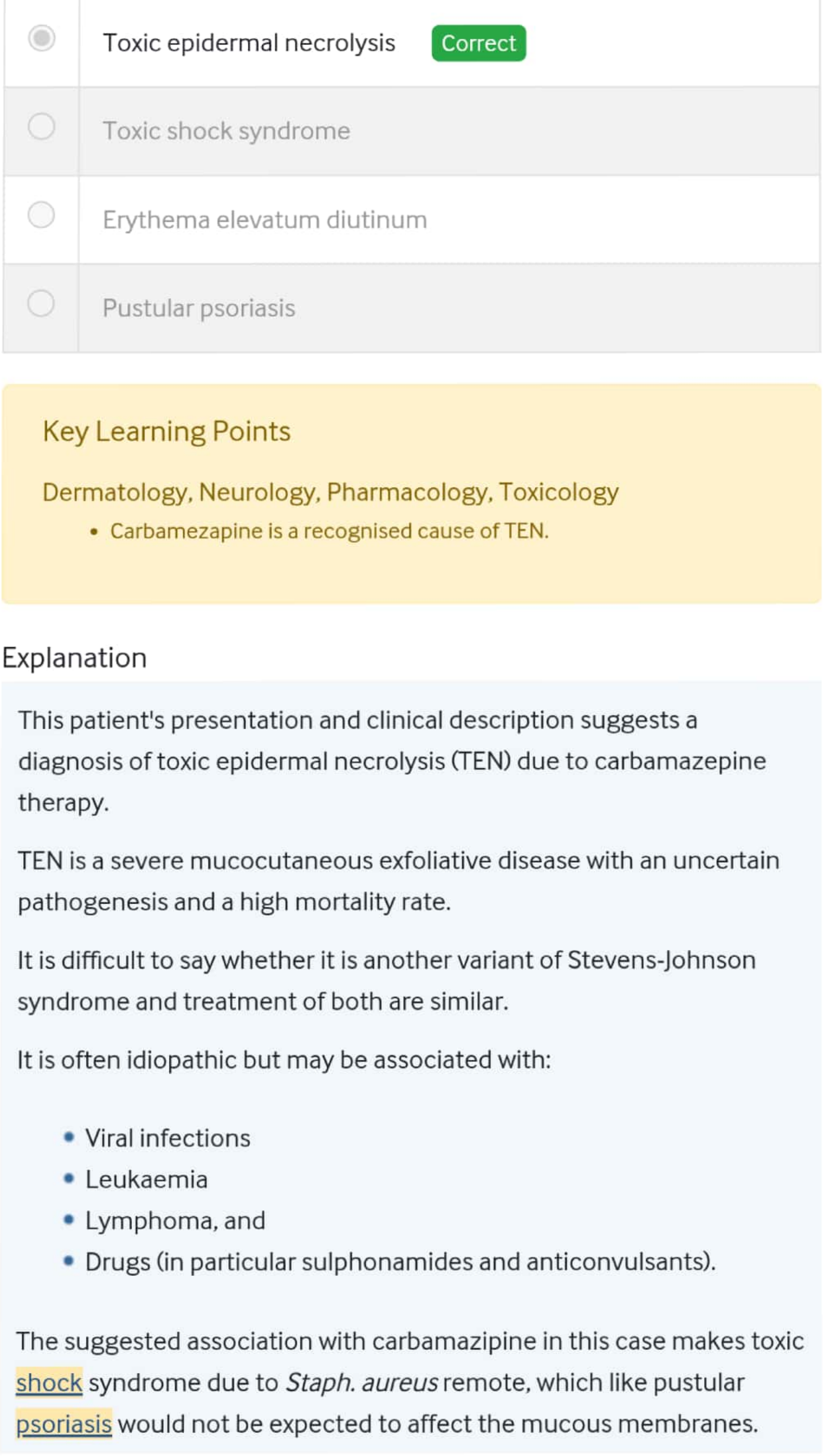
There is also blistering and inflammation of the oral cavity.

What is the likely diagnosis?

(Please select 1 option)

○ Exfoliative dermatitis
○ Toxic epidermal necrolysis
○ Toxic shock syndrome
○ Erythema elevatum diutinum
○ Pustular psoriasis

Submit Answer



● Toxic epidermal necrolysis **Correct**

○ Toxic shock syndrome

○ Erythema elevatum diutinum

○ Pustular psoriasis

Key Learning Points

Dermatology, Neurology, Pharmacology, Toxicology

- Carbamezapine is a recognised cause of TEN.

Explanation

This patient's presentation and clinical description suggests a diagnosis of toxic epidermal necrolysis (TEN) due to carbamazepine therapy.

TEN is a severe mucocutaneous exfoliative disease with an uncertain pathogenesis and a high mortality rate.

It is difficult to say whether it is another variant of Stevens-Johnson syndrome and treatment of both are similar.

It is often idiopathic but may be associated with:

- Viral infections
- Leukaemia
- Lymphoma, and
- Drugs (in particular sulphonamides and anticonvulsants).

The suggested association with carbamazipine in this case makes toxic shock syndrome due to *Staph. aureus* remote, which like pustular psoriasis would not be expected to affect the mucous membranes.

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A 22-year-old female presents with an eight weeks history of headaches, which have deteriorated over the last week.

She is aware of a frontal headache, which is constant in nature and unrelieved by paracetamol. These headaches have tended to be present when she wakes in the morning and lately have been present all day. Over the last six months she also explains that she has gained more than 7 kg in weight.

On examination, she is rather tearful, has a BMI of 32 kg/m^2 but has no nuchal rigidity and neurological examination is normal except for bilateral optic disc swelling on fundal examination. Her blood pressure is noted to be 122/88 mmHg and her temperature is 37°C.

Which of the following is the single best investigation to make the diagnosis?

(Please select 1 option)

○ EEG

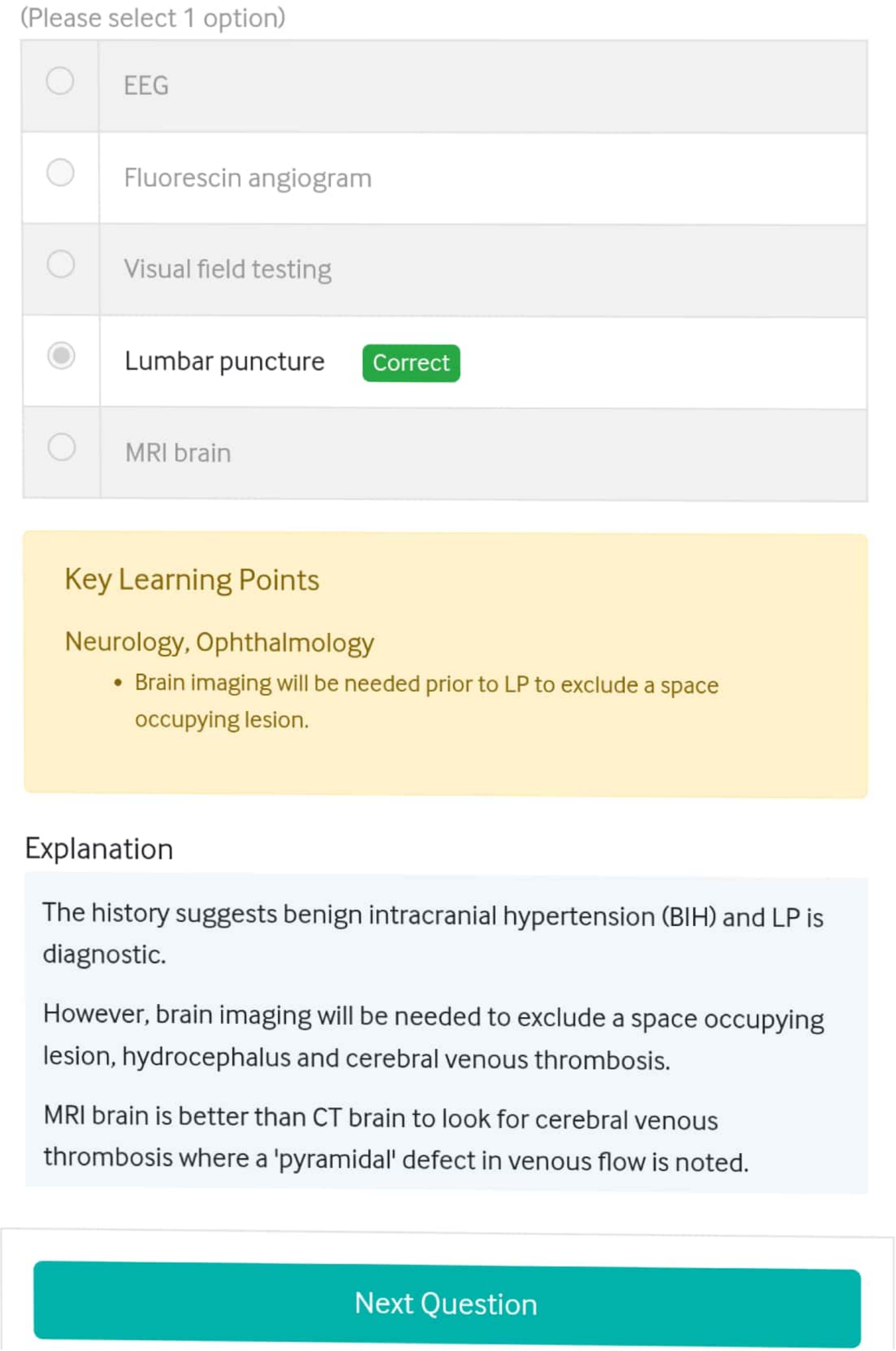
○ Fluorescin angiogram

○ Visual field testing

○ Lumbar puncture

○ MRI brain

Submit Answer



(Please select 1 option)

○ EEG

○ Fluorescin angiogram

○ Visual field testing

● Lumbar puncture **Correct**

○ MRI brain

Key Learning Points

Neurology, Ophthalmology

- Brain imaging will be needed prior to LP to exclude a space occupying lesion.

Explanation

The history suggests benign intracranial hypertension (BIH) and LP is diagnostic.

However, brain imaging will be needed to exclude a space occupying lesion, hydrocephalus and cerebral venous thrombosis.

MRI brain is better than CT brain to look for cerebral venous thrombosis where a 'pyramidal' defect in venous flow is noted.

Question 32 of 129

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You are referred a 40-year-old woman in a psychiatry ward who has a long history of schizophrenia resulting in multiple hospitalisations in the past.

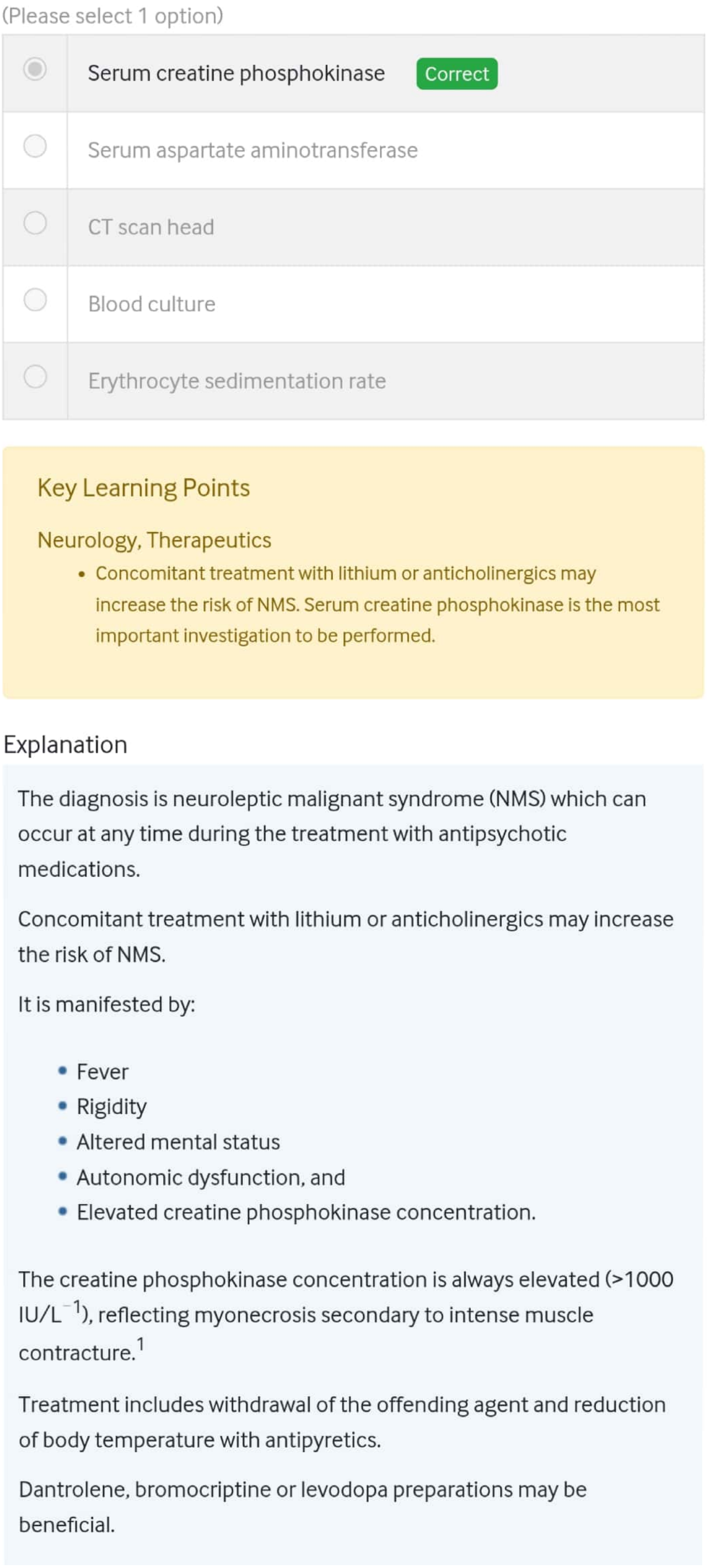
She gives a several week history of feeling generally unwell associated with increasing stiffness affecting the jaws and arms. Over the last few years she has been started on haloperidol with good symptom control. Her temperature was 38.5°C and BP 175/85 mmHg.

Which of the following is the most important investigation to be performed?

(Please select 1 option)

- ○ Serum creatine phosphokinase
- ○ Serum aspartate aminotransferase
- ○ CT scan head
- ○ Blood culture
- ○ Erythrocyte sedimentation rate

Submit Answer


(Please select 1 option)

●	Serum creatine phosphokinase **Correct**
○	Serum aspartate aminotransferase
○	CT scan head
○	Blood culture
○	Erythrocyte sedimentation rate

Key Learning Points

Neurology, Therapeutics

- Concomitant treatment with lithium or anticholinergics may increase the risk of NMS. Serum creatine phosphokinase is the most important investigation to be performed.

Explanation

The diagnosis is neuroleptic malignant syndrome (NMS) which can occur at any time during the treatment with antipsychotic medications.

Concomitant treatment with lithium or anticholinergics may increase the risk of NMS.

It is manifested by:

- Fever
- Rigidity
- Altered mental status
- Autonomic dysfunction, and
- Elevated creatine phosphokinase concentration.

The creatine phosphokinase concentration is always elevated (>1000 IU/L^{-1}), reflecting myonecrosis secondary to intense muscle contracture.[1]

Treatment includes withdrawal of the offending agent and reduction of body temperature with antipyretics.

Dantrolene, bromocriptine or levodopa preparations may be beneficial.

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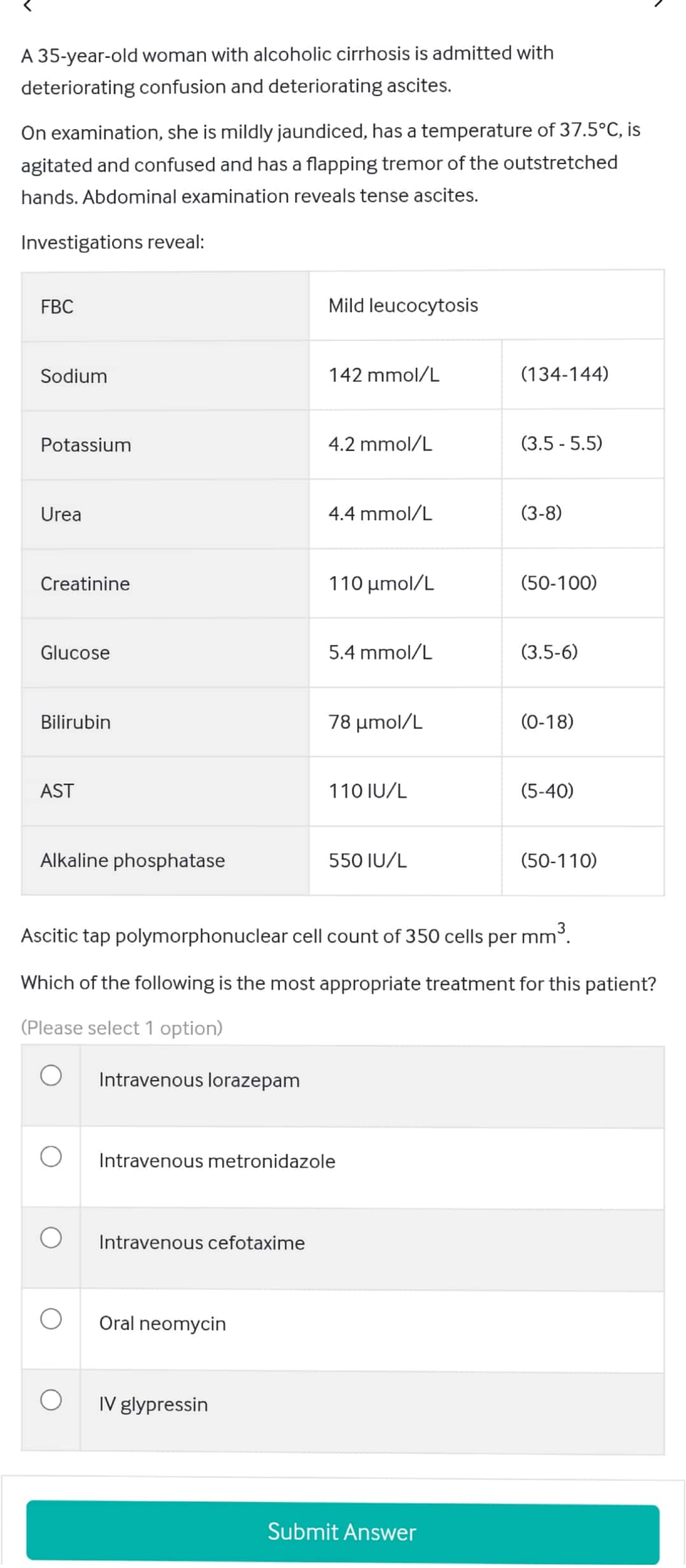

A 35-year-old woman with alcoholic cirrhosis is admitted with deteriorating confusion and deteriorating ascites.

On examination, she is mildly jaundiced, has a temperature of 37.5°C, is agitated and confused and has a flapping tremor of the outstretched hands. Abdominal examination reveals tense ascites.

Investigations reveal:

FBC	Mild leucocytosis	
Sodium	142 mmol/L	(134-144)
Potassium	4.2 mmol/L	(3.5 - 5.5)
Urea	4.4 mmol/L	(3-8)
Creatinine	110 µmol/L	(50-100)
Glucose	5.4 mmol/L	(3.5-6)
Bilirubin	78 µmol/L	(0-18)
AST	110 IU/L	(5-40)
Alkaline phosphatase	550 IU/L	(50-110)

Ascitic tap polymorphonuclear cell count of 350 cells per mm^3.

Which of the following is the most appropriate treatment for this patient?

(Please select 1 option)

- ○ Intravenous lorazepam
- ○ Intravenous metronidazole
- ○ Intravenous cefotaxime
- ○ Oral neomycin
- ○ IV glypressin

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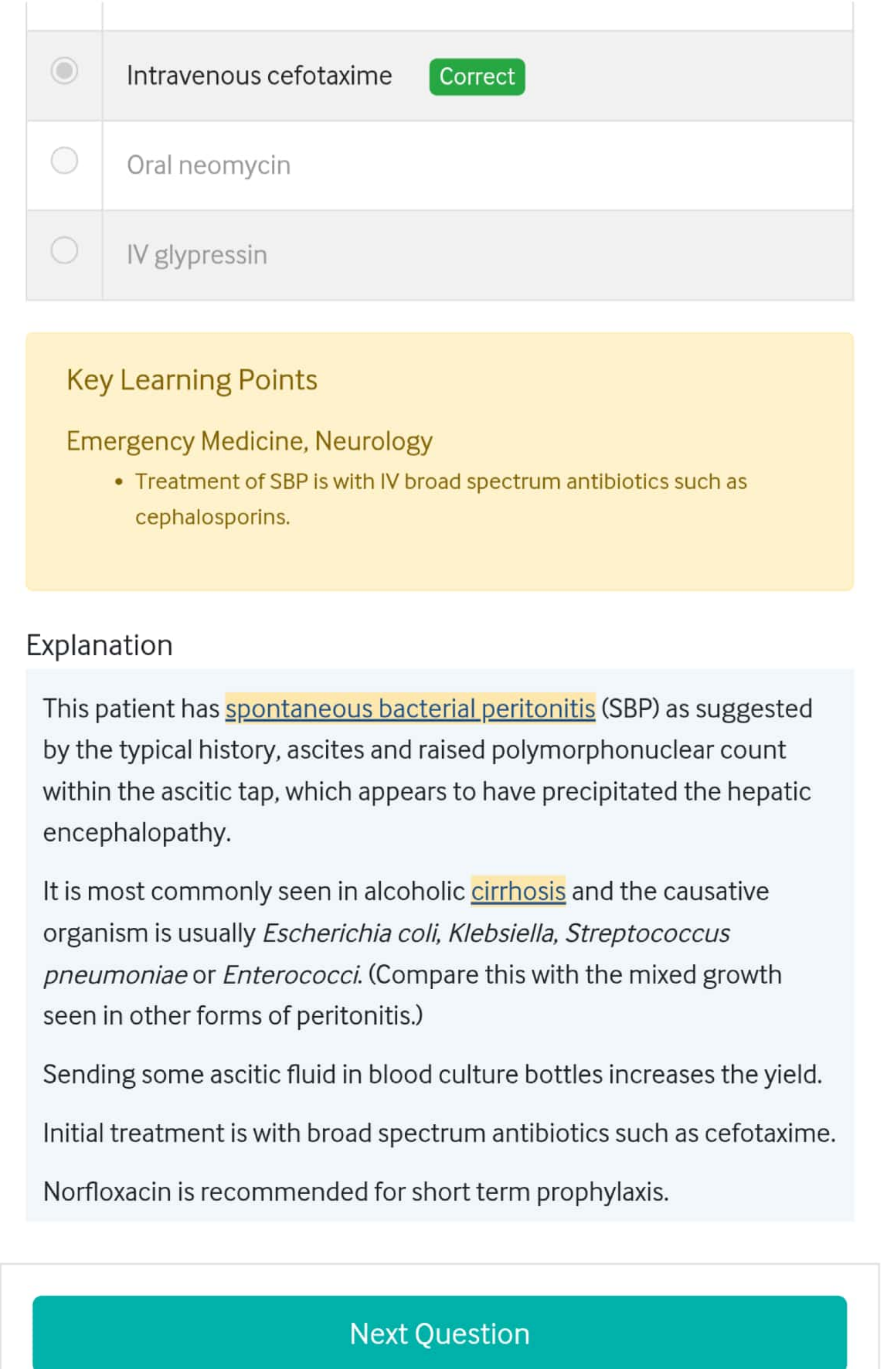

● Intravenous cefotaxime **Correct**

○ Oral neomycin

○ IV glypressin

Key Learning Points

Emergency Medicine, Neurology

- Treatment of SBP is with IV broad spectrum antibiotics such as cephalosporins.

Explanation

This patient has spontaneous bacterial peritonitis (SBP) as suggested by the typical history, ascites and raised polymorphonuclear count within the ascitic tap, which appears to have precipitated the hepatic encephalopathy.

It is most commonly seen in alcoholic cirrhosis and the causative organism is usually *Escherichia coli, Klebsiella, Streptococcus pneumoniae* or *Enterococci*. (Compare this with the mixed growth seen in other forms of peritonitis.)

Sending some ascitic fluid in blood culture bottles increases the yield.

Initial treatment is with broad spectrum antibiotics such as cefotaxime.

Norfloxacin is recommended for short term prophylaxis.

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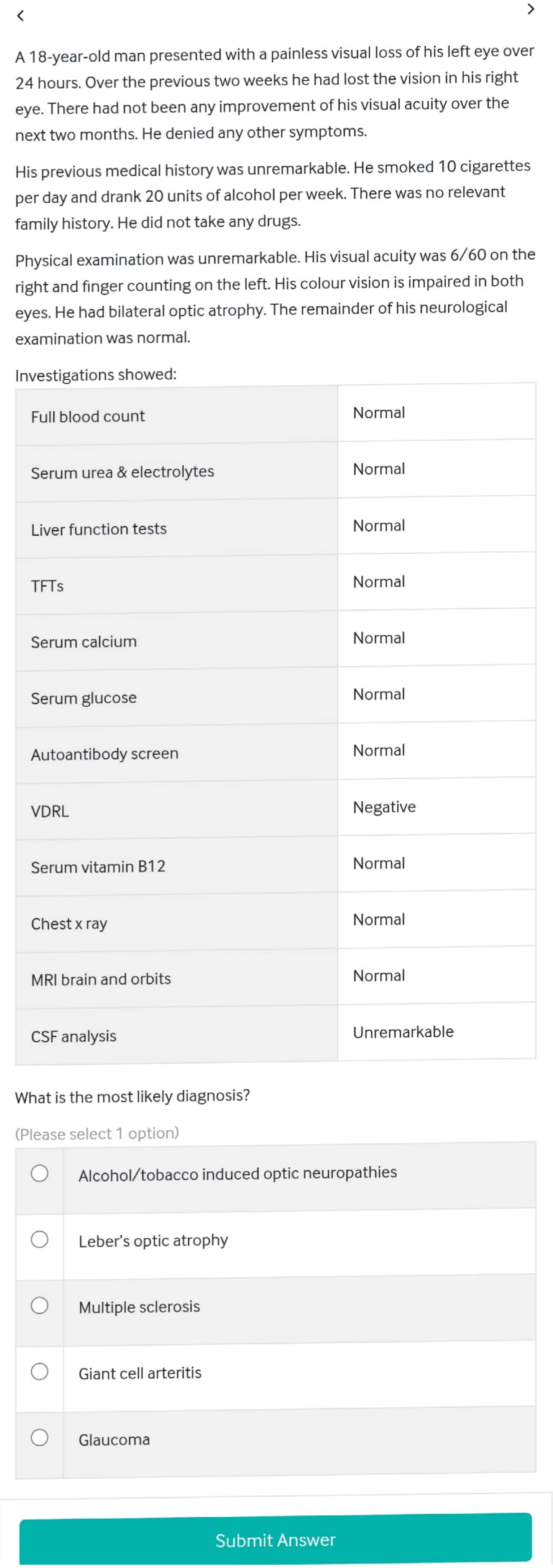


A 18-year-old man presented with a painless visual loss of his left eye over 24 hours. Over the previous two weeks he had lost the vision in his right eye. There had not been any improvement of his visual acuity over the next two months. He denied any other symptoms.

His previous medical history was unremarkable. He smoked 10 cigarettes per day and drank 20 units of alcohol per week. There was no relevant family history. He did not take any drugs.

Physical examination was unremarkable. His visual acuity was 6/60 on the right and finger counting on the left. His colour vision is impaired in both eyes. He had bilateral optic atrophy. The remainder of his neurological examination was normal.

Investigations showed:

Full blood count	Normal
Serum urea & electrolytes	Normal
Liver function tests	Normal
TFTs	Normal
Serum calcium	Normal
Serum glucose	Normal
Autoantibody screen	Normal
VDRL	Negative
Serum vitamin B12	Normal
Chest x ray	Normal
MRI brain and orbits	Normal
CSF analysis	Unremarkable

What is the most likely diagnosis?

(Please select 1 option)

- ○ Alcohol/tobacco induced optic neuropathies
- ○ Leber's optic atrophy
- ○ Multiple sclerosis
- ○ Giant cell arteritis
- ○ Glaucoma

○ Leber’s optic atrophy **This is the correct answer**

○ Multiple sclerosis

○ Giant cell arteritis

○ Glaucoma

Key Learning Points

Neurology

- Leber's optic atrophy usually affects young men and causes sequential painless optic neuropathies in days to weeks.

Explanation

Leber's optic atrophy usually affects young men. It causes sequential optic neuropathies in days to weeks. It is typically painless and severe. Visual acuity fails to improve.

Mutations in the MT-ND1, MT-ND4, MT-ND4L, and MT-ND6 genes cause Leber hereditary optic atrophy. These genes are contained in mitochondrial DNA. A significant percentage of people with a mutation that causes Leber hereditary optic atrophy do not develop any features of the disorder. Specifically, more than 50% of males with a mutation and more than 85% of females with a mutation never experience vision loss or related medical problems.

Optic neuritis is usually painful, and visual acuity improves over a matter of weeks.

Giant cell arteritis affects elderly patients.

Alcohol/tobacco optic neuropathies are usually chronic.

Next Question

CamScanner الممسوحة ضوئيا بـ

A 70-year-old woman is admitted to Emergency department following a road traffic accident 3 hours ago.

She is maintaining her own airway with a respiratory rate of 18 breaths per minute and SpO_2 on air is 97%. Her heart rate is 85 beats per minute and her blood pressure is 140/70 mmHg. She has a visible bruise over the right temporal region and a Glasgow coma score (GCS) of 14. There are no other apparent injuries.

She has a past medical history of hypertension and atrial fibrillation, her normal medications include atenolol and warfarin. She does not recall the accident. A radiology service is available on site but the nearest neurosurgical unit is 40 miles away.

What is the next appropriate management option?

(Please select 1 option)

○ Transfer to the neurosurgical unit for neurological observations

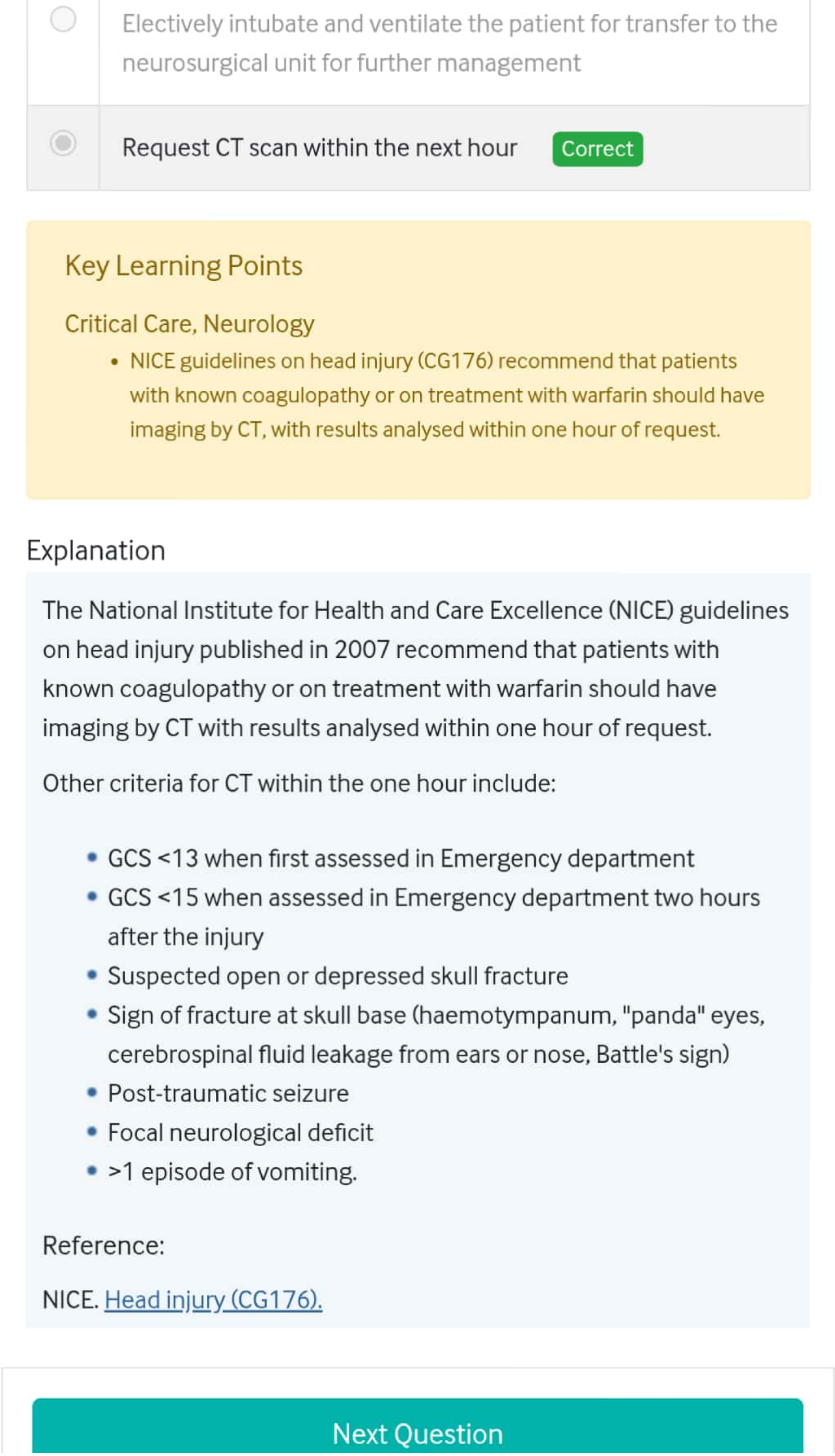
○ Request CT scan within the next eight hours

○ Admit to the Emergency department ward for neurological observations

○ Electively intubate and ventilate the patient for transfer to the neurosurgical unit for further management

○ Request CT scan within the next hour

Submit Answer



○	Electively intubate and ventilate the patient for transfer to the neurosurgical unit for further management
●	Request CT scan within the next hour **Correct**

Key Learning Points

Critical Care, Neurology

- NICE guidelines on head injury (CG176) recommend that patients with known coagulopathy or on treatment with warfarin should have imaging by CT, with results analysed within one hour of request.

Explanation

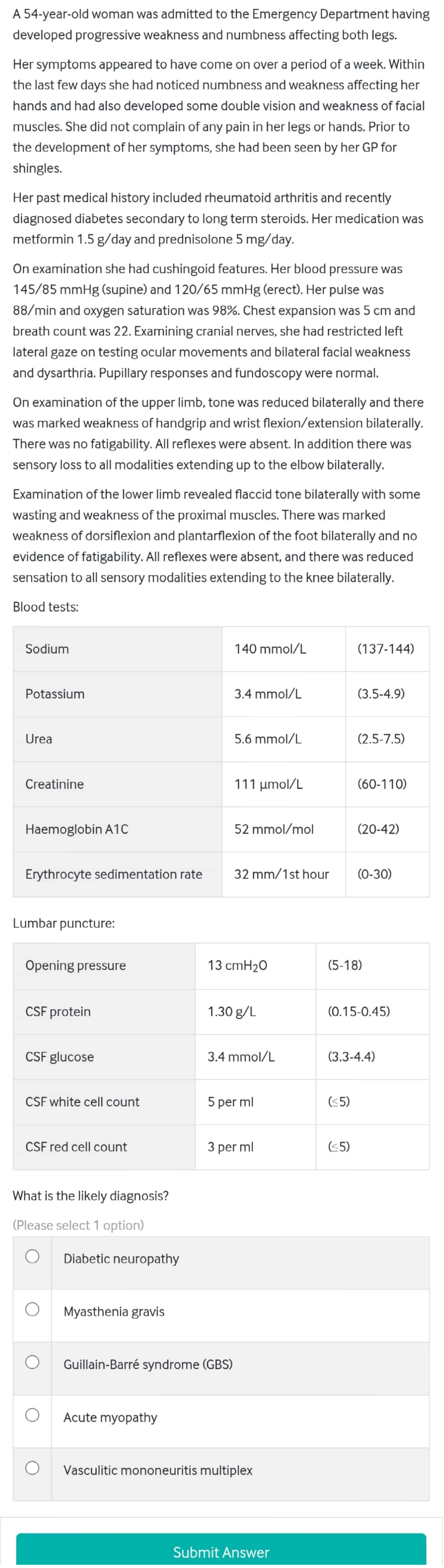
The National Institute for Health and Care Excellence (NICE) guidelines on head injury published in 2007 recommend that patients with known coagulopathy or on treatment with warfarin should have imaging by CT with results analysed within one hour of request.

Other criteria for CT within the one hour include:

- GCS <13 when first assessed in Emergency department
- GCS <15 when assessed in Emergency department two hours after the injury
- Suspected open or depressed skull fracture
- Sign of fracture at skull base (haemotympanum, "panda" eyes, cerebrospinal fluid leakage from ears or nose, Battle's sign)
- Post-traumatic seizure
- Focal neurological deficit
- >1 episode of vomiting.

Reference:

NICE. Head injury (CG176).



A 54-year-old woman was admitted to the Emergency Department having developed progressive weakness and numbness affecting both legs.

Her symptoms appeared to have come on over a period of a week. Within the last few days she had noticed numbness and weakness affecting her hands and had also developed some double vision and weakness of facial muscles. She did not complain of any pain in her legs or hands. Prior to the development of her symptoms, she had been seen by her GP for shingles.

Her past medical history included rheumatoid arthritis and recently diagnosed diabetes secondary to long term steroids. Her medication was metformin 1.5 g/day and prednisolone 5 mg/day.

On examination she had cushingoid features. Her blood pressure was 145/85 mmHg (supine) and 120/65 mmHg (erect). Her pulse was 88/min and oxygen saturation was 98%. Chest expansion was 5 cm and breath count was 22. Examining cranial nerves, she had restricted left lateral gaze on testing ocular movements and bilateral facial weakness and dysarthria. Pupillary responses and fundoscopy were normal.

On examination of the upper limb, tone was reduced bilaterally and there was marked weakness of handgrip and wrist flexion/extension bilaterally. There was no fatigability. All reflexes were absent. In addition there was sensory loss to all modalities extending up to the elbow bilaterally.

Examination of the lower limb revealed flaccid tone bilaterally with some wasting and weakness of the proximal muscles. There was marked weakness of dorsiflexion and plantarflexion of the foot bilaterally and no evidence of fatigability. All reflexes were absent, and there was reduced sensation to all sensory modalities extending to the knee bilaterally.

Blood tests:

Sodium	140 mmol/L	(137-144)
Potassium	3.4 mmol/L	(3.5-4.9)
Urea	5.6 mmol/L	(2.5-7.5)
Creatinine	111 µmol/L	(60-110)
Haemoglobin A1C	52 mmol/mol	(20-42)
Erythrocyte sedimentation rate	32 mm/1st hour	(0-30)

Lumbar puncture:

Opening pressure	13 cmH_2O	(5-18)
CSF protein	1.30 g/L	(0.15-0.45)
CSF glucose	3.4 mmol/L	(3.3-4.4)
CSF white cell count	5 per ml	(≤5)
CSF red cell count	3 per ml	(≤5)

What is the likely diagnosis?

(Please select 1 option)

○ Diabetic neuropathy

○ Myasthenia gravis

○ Guillain-Barré syndrome (GBS)

○ Acute myopathy

○ Vasculitic mononeuritis multiplex

● Guillain-Barré syndrome (GBS) **Correct**

○ Acute myopathy

○ Vasculitic mononeuritis multiplex

Key Learning Points

Neurology

- GBS presents with a short history of ascending symmetrical progressive weakness and numbness affecting initially the legs and extending to involve the upper limb.

Explanation

This patient presents with a short history of ascending symmetrical progressive weakness and numbness affecting initially the legs and extending to involve the upper limb.

In addition she has evidence of autonomic dysfunction demonstrated by postural hypotension.

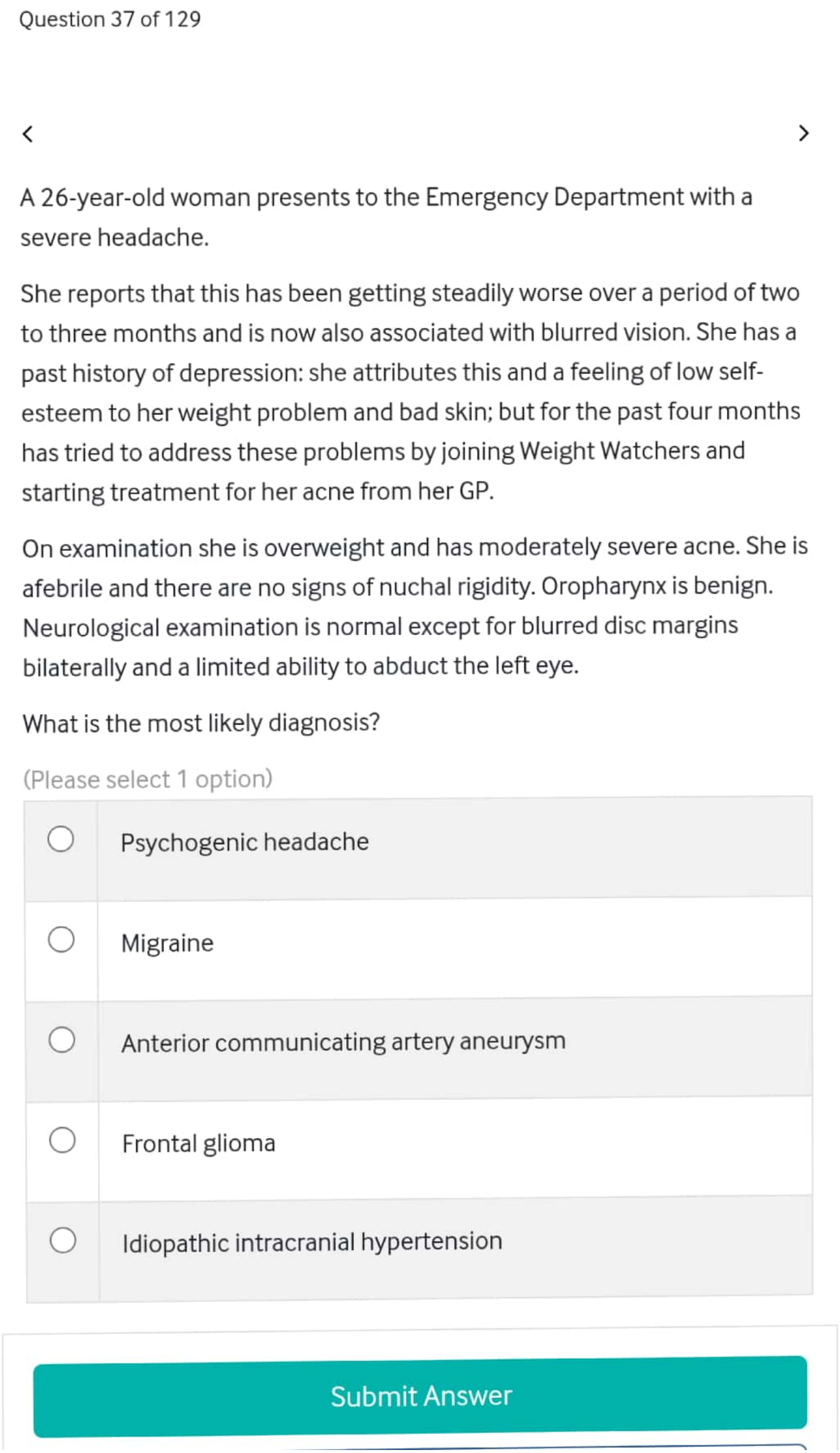
The diagnosis is Guillain-Barré syndrome (GBS).

Other clues include prior varicella zoster viral infection and elevated cerebrospinal fluid (CSF) protein in absence of a pleocytosis.

This patient probably has in addition to GBS a steroid myopathy affecting her legs, which is probably long term in view of muscle wasting. This patient does not have fatigable weakness found in myasthenia gravis and her reflexes and sensory examination would be normal.

A diabetic neuropathy with cranial nerve involvement is unlikely, as she has only recently been diagnosed with diabetes, and has only slight elevation of her HbA_{1c}.

This is not a clinical picture for vasculitic mononeuritis multiplex which is rare, and where specific individual peripheral nerves are targeted.



Question 37 of 129

A 26-year-old woman presents to the Emergency Department with a severe headache.

She reports that this has been getting steadily worse over a period of two to three months and is now also associated with blurred vision. She has a past history of depression: she attributes this and a feeling of low self-esteem to her weight problem and bad skin; but for the past four months has tried to address these problems by joining Weight Watchers and starting treatment for her acne from her GP.

On examination she is overweight and has moderately severe acne. She is afebrile and there are no signs of nuchal rigidity. Oropharynx is benign. Neurological examination is normal except for blurred disc margins bilaterally and a limited ability to abduct the left eye.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Psychogenic headache
- ○ Migraine
- ○ Anterior communicating artery aneurysm
- ○ Frontal glioma
- ○ Idiopathic intracranial hypertension

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○ Idiopathic intracranial hypertension **Correct**

Key Learning Points

Neurology

- Idiopathic intracranial hypertension is more commonly associated with obesity, use of the oral contraceptive/tetracycline and pregnancy.

Explanation

This is a typical history and presentation of idiopathic intracranial hypertension (IIH; previously known as benign intracranial hypertension; pseudotumour cerebri).

IIH typically affects young obese women.

Other risk factors include:

- The oral contraceptive pill
- Treatments for acne (tetracycline, nitrofurantoin, retinoids), and
- Hypervitaminosis A.

Presentation is with a headache, which may be severe.

There may be loss of peripheral vision and impaired visual acuity if papilloedema is severe. Reduction in colour vision is common and a significant number of patients develop a CN VI palsy.

CT scan is often normal; the diagnosis is confirmed by finding an elevated CSF opening pressure (more than 20 cm H_2O).

CSF protein, glucose and cell count will be normal.

Further Reading

Medscape. Idiopathic Intracranial Hypertension.

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A 45-year-old man presented to his GP with a six day history of sudden onset left sided blurred vision. In addition he had noticed he was bumping into things on his left and this had led to a number of falls. He had not experienced any pain in the left eye or had any associated headache. He had not noticed any weakness or sensory disturbance in his arms or legs, and his bladder and bowels were functioning normally.

He had no known medical conditions, but was being investigated for an isolated seizure and also some memory difficulties. He was a non-smoker and worked on a cattle farm.

On examination, he had yellowish papules on the neck. His blood pressure was 140/80 mmHg, pulse was 72 beats per minute and regular and heart sounds were normal. Corrected visual acuity was 6/9 in the left eye and 6/6 in the right. There was a left relative afferent pupillary defect. The intraocular pressure was normal in both eyes. Fundoscopy revealed some pallor of the left optic disc and also angioid streaks. Visual field testing revealed a left homonymous hemianopia. The rest of the cranial nerve examination together with the peripheral nervous system examination was normal.

Investigations revealed:

Sodium	135 mmol/L	(137-144)
Potassium	3.9 mmol/L	(3.5-4.9)
Urea	4.5 mmol/L	(2.5-7.5)
Creatinine	90 μmol/L	(60-110)
Glucose	5.2 mmol/L	(3.0-6.0)
Cholesterol	4.6 mmo/L	(<5.2)
Erythrocyte sedimentation rate	15 mm	(0-15)

A transthoracic echocardiogram was normal.

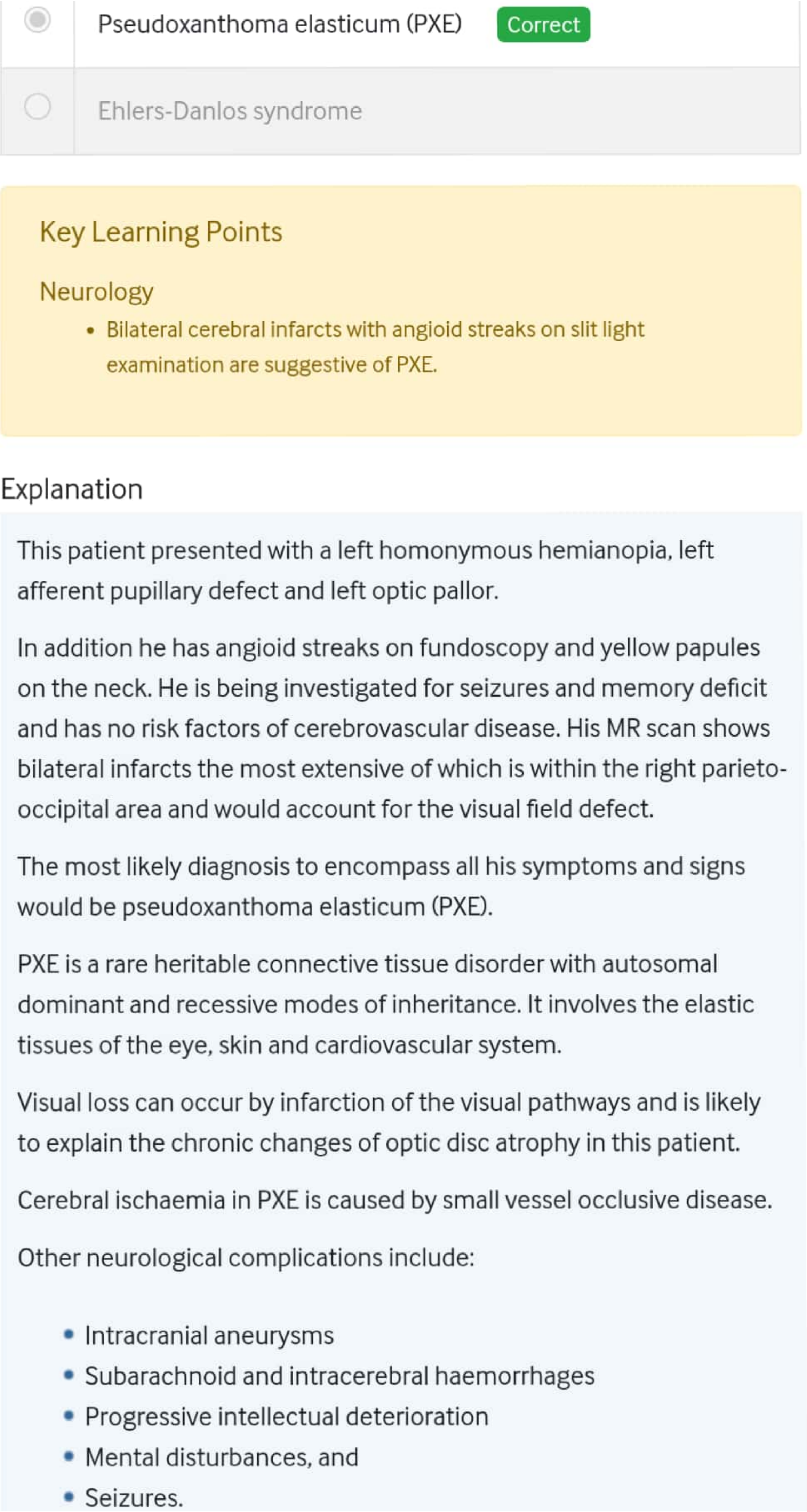
An MRI brain and orbits (FLAIR) showed multiple bilateral ischaemic infarcts in the brain and a large right parieto-occipital infarct. The orbits and optic nerves were normal.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Multiple sclerosis
- ○ Pituitary tumour
- ○ Giant cell arteritis
- ○ Pseudoxanthoma elasticum (PXE)
- ○ Ehlers-Danlos syndrome

Submit Answer



● Pseudoxanthoma elasticum (PXE)

○ Ehlers-Danlos syndrome

Key Learning Points

Neurology

- Bilateral cerebral infarcts with angioid streaks on slit light examination are suggestive of PXE.

Explanation

This patient presented with a left homonymous hemianopia, left afferent pupillary defect and left optic pallor.

In addition he has angioid streaks on fundoscopy and yellow papules on the neck. He is being investigated for seizures and memory deficit and has no risk factors of cerebrovascular disease. His MR scan shows bilateral infarcts the most extensive of which is within the right parieto-occipital area and would account for the visual field defect.

The most likely diagnosis to encompass all his symptoms and signs would be pseudoxanthoma elasticum (PXE).

PXE is a rare heritable connective tissue disorder with autosomal dominant and recessive modes of inheritance. It involves the elastic tissues of the eye, skin and cardiovascular system.

Visual loss can occur by infarction of the visual pathways and is likely to explain the chronic changes of optic disc atrophy in this patient.

Cerebral ischaemia in PXE is caused by small vessel occlusive disease.

Other neurological complications include:

- Intracranial aneurysms
- Subarachnoid and intracerebral haemorrhages
- Progressive intellectual deterioration
- Mental disturbances, and
- Seizures.

A 65-year-old female is admitted with an intracranial bleed under the care of the neurosurgeons.

Following magnetic resonance angiography she undergoes clipping of a cerebral arterial aneurysm and was well the following morning.

The surgical team document the following blood chemistry results on successive postoperative days:

	Plasma Sodium	Potassium	Urea	Creatinine
Day 1	130 mmol/L	3.5 mmol/L	4.2 mmol/L	95 µmol/L
Day 2	127 mmol/L	3.4 mmol/L	4.2 mmol/L	90 µmol/L
Day 3	124 mmol/L	3.4 mmol/L	4.4 mmol/L	76 µmol/L
Day 4	120 mmol/L	3.5 mmol/L	5.0 mmol/L	70 µmol/L

Normal Ranges:

Plasma sodium	137-144 mmol/L
Potassium	3.5-4.9 mmol/L
Urea	2.5-7.5 mmol/L
Creatinine	60-110 µmol/L

On day four she was commenced on a fluid restriction of 1 litre per day. Investigations at that time show:

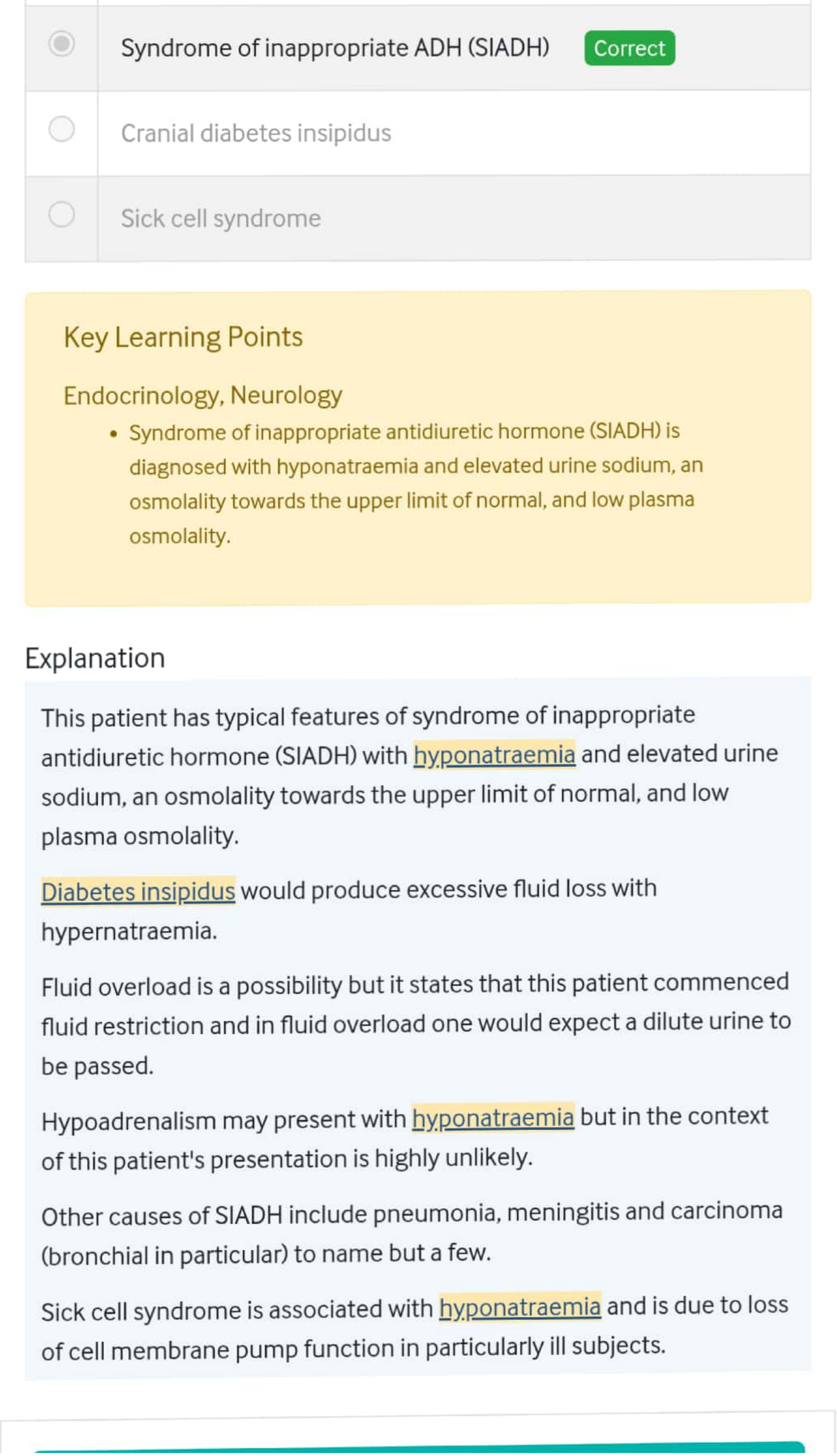
Plasma osmolality	262 mOsmol/L	(278-305)
Urine osmolality	700 mOsmol/L	(350-1000)
Urine sodium	70 mmol/L	-

What diagnosis would most likely explain these findings?

(Please select 1 option)

- ○ Fluid overload
- ○ Hypoadrenalism
- ○ Syndrome of inappropriate ADH (SIADH)
- ○ Cranial diabetes insipidus
- ○ Sick cell syndrome

Submit Answer



● Syndrome of inappropriate ADH (SIADH) Correct

○ Cranial diabetes insipidus

○ Sick cell syndrome

Key Learning Points

Endocrinology, Neurology

- Syndrome of inappropriate antidiuretic hormone (SIADH) is diagnosed with hyponatraemia and elevated urine sodium, an osmolality towards the upper limit of normal, and low plasma osmolality.

Explanation

This patient has typical features of syndrome of inappropriate antidiuretic hormone (SIADH) with hyponatraemia and elevated urine sodium, an osmolality towards the upper limit of normal, and low plasma osmolality.

Diabetes insipidus would produce excessive fluid loss with hypernatraemia.

Fluid overload is a possibility but it states that this patient commenced fluid restriction and in fluid overload one would expect a dilute urine to be passed.

Hypoadrenalism may present with hyponatraemia but in the context of this patient's presentation is highly unlikely.

Other causes of SIADH include pneumonia, meningitis and carcinoma (bronchial in particular) to name but a few.

Sick cell syndrome is associated with hyponatraemia and is due to loss of cell membrane pump function in particularly ill subjects.

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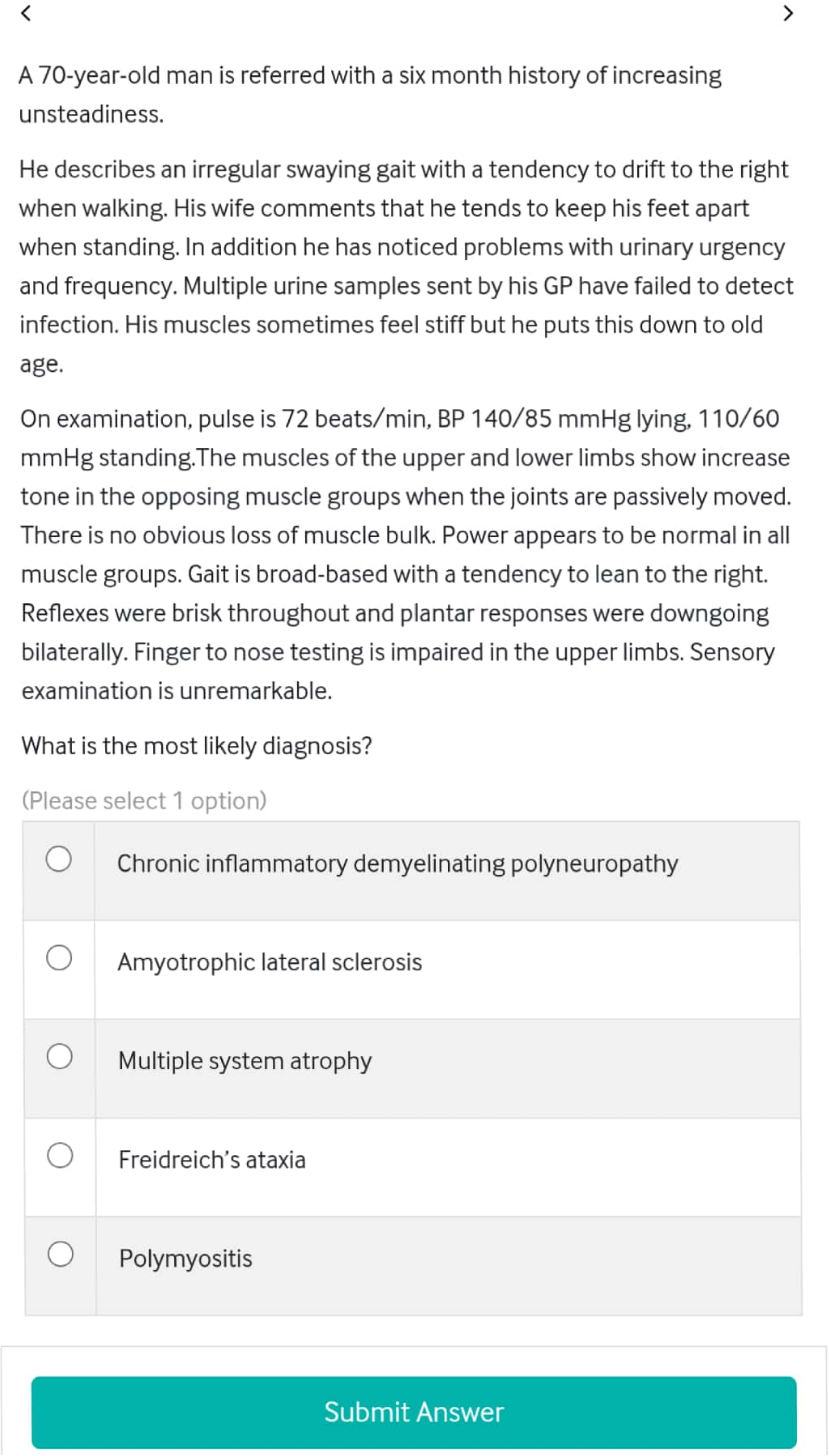


A 70-year-old man is referred with a six month history of increasing unsteadiness.

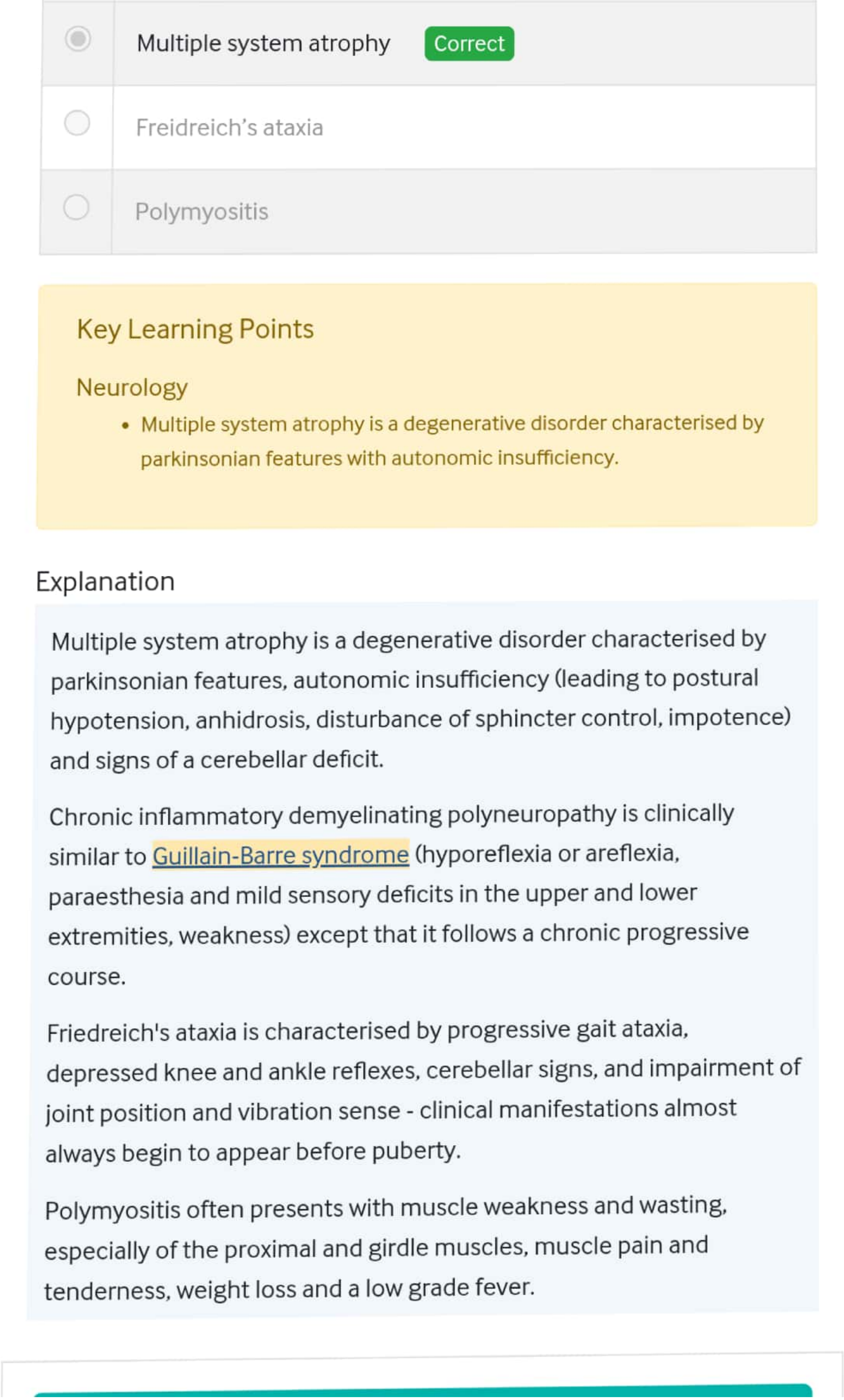
He describes an irregular swaying gait with a tendency to drift to the right when walking. His wife comments that he tends to keep his feet apart when standing. In addition he has noticed problems with urinary urgency and frequency. Multiple urine samples sent by his GP have failed to detect infection. His muscles sometimes feel stiff but he puts this down to old age.

On examination, pulse is 72 beats/min, BP 140/85 mmHg lying, 110/60 mmHg standing.The muscles of the upper and lower limbs show increase tone in the opposing muscle groups when the joints are passively moved. There is no obvious loss of muscle bulk. Power appears to be normal in all muscle groups. Gait is broad-based with a tendency to lean to the right. Reflexes were brisk throughout and plantar responses were downgoing bilaterally. Finger to nose testing is impaired in the upper limbs. Sensory examination is unremarkable.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Chronic inflammatory demyelinating polyneuropathy
- ○ Amyotrophic lateral sclerosis
- ○ Multiple system atrophy
- ○ Freidreich’s ataxia
- ○ Polymyositis



● Multiple system atrophy **Correct**

○ Freidreich's ataxia

○ Polymyositis

Key Learning Points

Neurology

- Multiple system atrophy is a degenerative disorder characterised by parkinsonian features with autonomic insufficiency.

Explanation

Multiple system atrophy is a degenerative disorder characterised by parkinsonian features, autonomic insufficiency (leading to postural hypotension, anhidrosis, disturbance of sphincter control, impotence) and signs of a cerebellar deficit.

Chronic inflammatory demyelinating polyneuropathy is clinically similar to Guillain-Barre syndrome (hyporeflexia or areflexia, paraesthesia and mild sensory deficits in the upper and lower extremities, weakness) except that it follows a chronic progressive course.

Friedreich's ataxia is characterised by progressive gait ataxia, depressed knee and ankle reflexes, cerebellar signs, and impairment of joint position and vibration sense - clinical manifestations almost always begin to appear before puberty.

Polymyositis often presents with muscle weakness and wasting, especially of the proximal and girdle muscles, muscle pain and tenderness, weight loss and a low grade fever.

A 32-year-old woman presented to the Emergency department with a sudden onset weakness affecting the left face, arm and leg. She had no disturbances of consciousness, sensation, vision or speech.

She had been complaining of headaches over several weeks, which appeared generalised and had also noticed in the last week some twitching which was affecting her left hand. She had recently been to her GP complaining of frequent epistaxis, otherwise she had no past medical history and took no regular medication.

She was a smoker of six cigarettes per day and drank 10 units of alcohol per week. Her mother died of gastrointestinal bleeding, which was thought secondary to alcohol abuse.

On examination there were punctiform lesions on oral inspection. Her blood pressure was 130/75 mmHg, pulse was 78 beats per minute and temperature was 35°C. Cardiovascular, chest and abdominal examinations were normal. There was left facial asymmetry with evidence of facial weakness. Pupils, visual fields and fundoscopy were normal.

On peripheral nervous system examination, there was evidence of left sided pyramidal weakness with hyper-reflexia and increased tone. Lower limb examination revealed left sided pyramidal weakness with brisk left knee reflex, ankle jerk and a left extensor plantar. There was also some left hemisensory loss.

Investigations revealed:

Haemoglobin	102 g/L	(130-180)
Mean cell volume	72 fL	(80-96)
White cell count	12.0×10^9/L	(4-11)
Platelets	345×10^9/L	(150-400)
Prothrombin time	13.5 secs	(11.5-15.5)
APTT	35 secs	(30-40)
Bleeding time	6 mins	(2-8)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	5.6 mmol/L	(2.5-7.5)
Serum creatinine	80 µmol/L	(60-110)
Fasting plasma glucose	5.6 mmol/L	(3.0-6.0)
Serum cholesterol	4.5 mmol/L	(< 5.2)

An MRI scan of the brain showed a right fronto-parietal haemorrhage.

Given the above history and investigations so far, what is the likely diagnosis?

(Please select 1 option)

○ Cerebral amyloid angiopathy

○ von Willebrand's disease

○ Autosomal dominant polycystic kidney disease

○ Intracranial aneurysm

○ Hereditary haemorrhagic telangectasia

Submit Answer

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○ Intracranial aneurysm

● Hereditary haemorrhagic telangectasia **Correct**

Key Learning Points

Neurology

- Hereditary haemorrhagic telangiectasia presents with epistaxis, recurrent haemoptysis and cerebral and pulmonary arteriovenous malformations.

Explanation

This patient presents with a haemorrhagic fronto-parietal stroke, most likely secondary to an arteriovenous malformation, previous headaches and simple partial seizures affecting the left hand.

She has a history of epistaxis, punctate lesions in her mouth and a clue that her mother had gastrointestinal bleeding the aetiology of which was unclear.

These clues point to a diagnosis of hereditary haemorrhagic telangectasia. This presents with epistaxis, recurrent haemoptysis and cerebral and pulmonary arteriovenous malformations.

There are two genetic types associated with mutations involving the endoglin gene located on chromosome 9q and activin receptor like kinase 1, located on 12q.

The history is not compatible with polycystic kidney disease, which is associated with intracerebral aneurysms.

Cerebral amyloid angiopathy can present with haemorrhagic strokes or dementia.

von Willebrand's disease is associated with mucosal bleeding and abnormal bleeding time.

Next Question

A 59-year-old female pub landlady presents with acute, severe lumbar back pain.

There is no history of orthopaedic problems and until this event she had been in perfect health.

The patient complains of paraesthesia in the lower limbs and on further questioning has not voided urine since the onset of the pain.

Neurological examination reveals weakness (3/5 of both lower limbs) and loss of sensation to touch in L4, L5 and S1. Vibration sensation and joint position sensation is preserved. Reflexes in the ankles and knees are absent and the plantar response is equivocal.

The blood pressure is 158/68 in her left arm and 150/62 in her right, heart rate 95 bpm, temperature 36.9°C and ECG shows normal sinus rhythm with no ischaemic changes.

The remainder of the examination is normal.

Which of the following investigations will be most useful in securing a diagnosis?

(Please select 1 option)

- ○ USS abdomen
- ○ MRI spinal cord
- ○ Duplex scan of aorta
- ○ Rectal examination
- ○ CT head

○ USS abdomen

● MRI spinal cord **Correct**

○ Duplex scan of aorta

○ Rectal examination

○ CT head

Key Learning Points

Neurology

- Clinical presentation of cauda equina syndrome is varied, but can include low back pain with unilateral or bilateral lower limb motor and/or sensory abnormality and bowel and/or bladder dysfunction with saddle and perineal anaesthesia.

Explanation

This patient has a number of features of cauda equina syndrome. There is distal weakness, with associated sensory loss. Whilst classically patients present with a sensory level, this is variable in clinical practice.

Causes of cauda equina syndrome include herniation of a lumbar disc (at L4/L5 and L5/S1), tumour (metastases, lymphoma, primary spinal tumours), trauma and infection (epidural abscess).

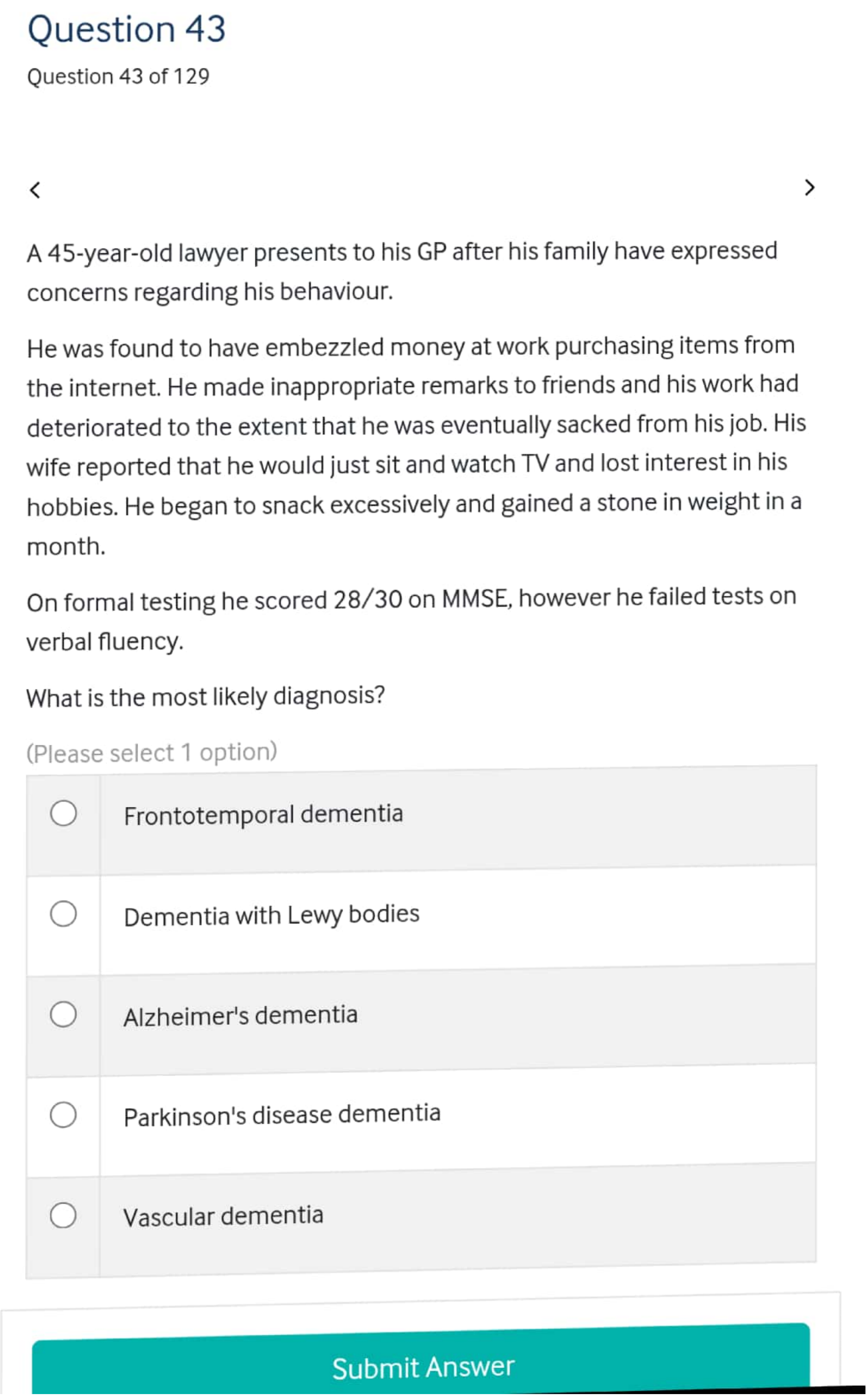
Clinical presentation is varied, but can include low back pain with unilateral or bilateral lower limb motor and/or sensory abnormality and bowel and/or bladder dysfunction with saddle and perineal anaesthesia.

MRI is the investigation of choice to confirm the diagnosis and determine the level of compression and underlying cause. Delayed diagnosis and intervention can lead to permanent neurological damage, and therefore MRI should be undertaken in a timely fashion.

Determining the presence of bowel dysfunction (with reduced anal tone and sensation) can be helpful prognostically, but does not assist with the differential diagnosis.

The pattern of weakness is not consistent with an intracranial cause, and USS abdomen or duplex scan of the aorta are unlikely to help here.

Next Question



Question 43

Question 43 of 129

A 45-year-old lawyer presents to his GP after his family have expressed concerns regarding his behaviour.

He was found to have embezzled money at work purchasing items from the internet. He made inappropriate remarks to friends and his work had deteriorated to the extent that he was eventually sacked from his job. His wife reported that he would just sit and watch TV and lost interest in his hobbies. He began to snack excessively and gained a stone in weight in a month.

On formal testing he scored 28/30 on MMSE, however he failed tests on verbal fluency.

What is the most likely diagnosis?

(Please select 1 option)

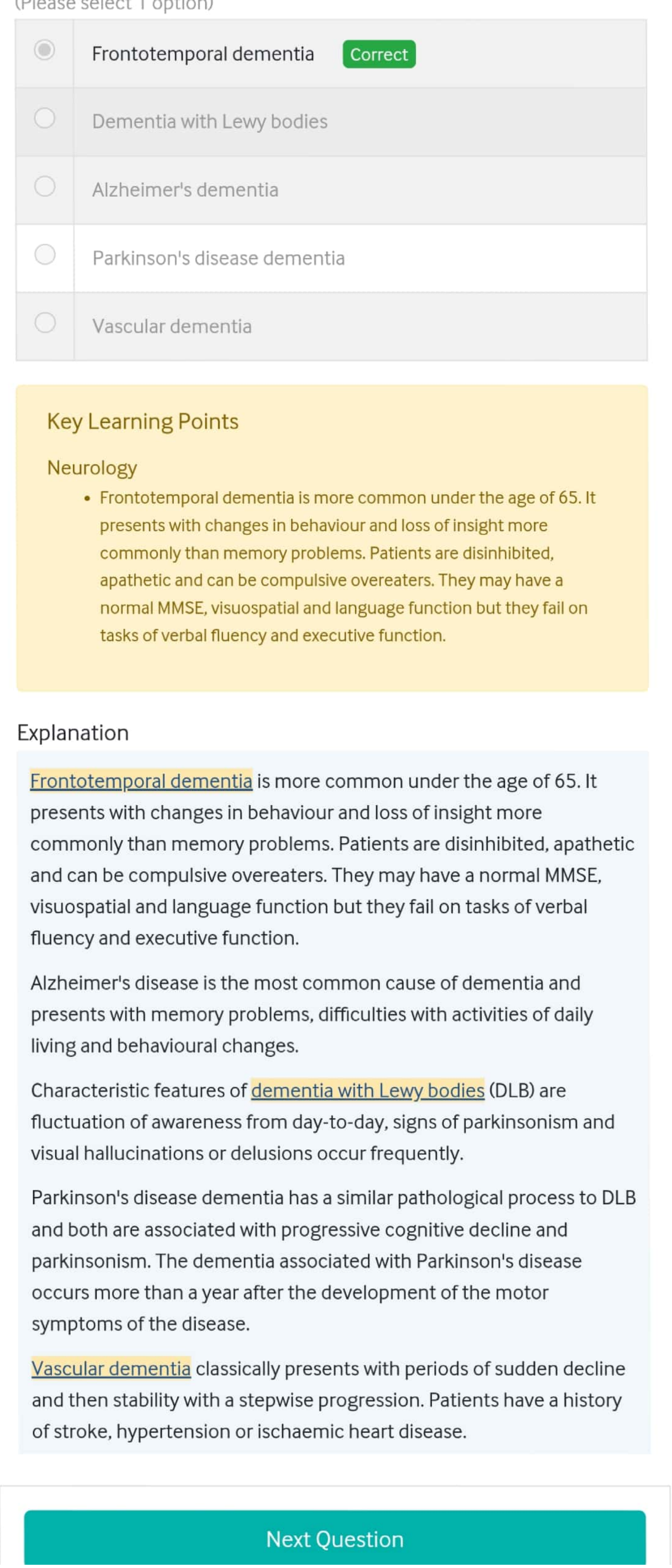
○ Frontotemporal dementia

○ Dementia with Lewy bodies

○ Alzheimer's dementia

○ Parkinson's disease dementia

○ Vascular dementia



(Please select 1 option)

● Frontotemporal dementia

○ Dementia with Lewy bodies

○ Alzheimer's dementia

○ Parkinson's disease dementia

○ Vascular dementia

Key Learning Points

Neurology

- Frontotemporal dementia is more common under the age of 65. It presents with changes in behaviour and loss of insight more commonly than memory problems. Patients are disinhibited, apathetic and can be compulsive overeaters. They may have a normal MMSE, visuospatial and language function but they fail on tasks of verbal fluency and executive function.

Explanation

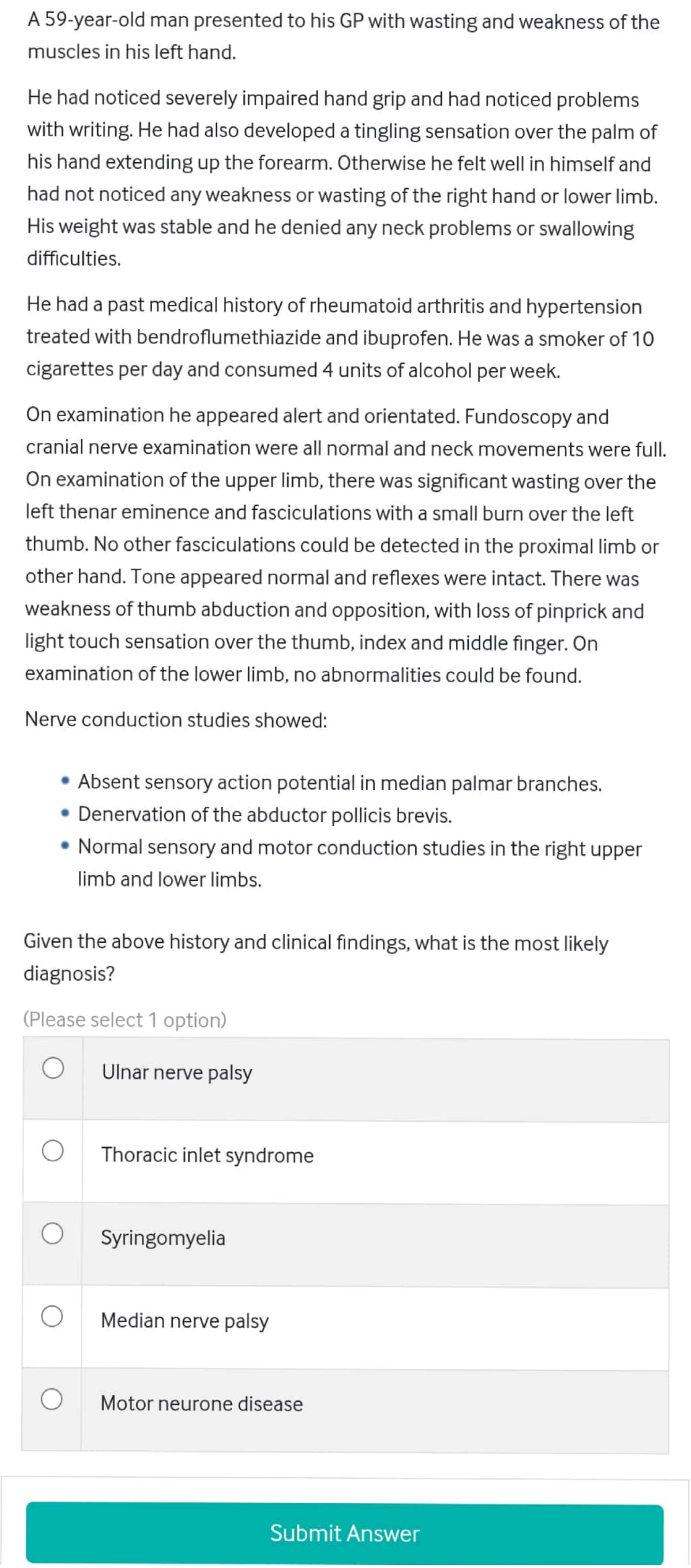
Frontotemporal dementia is more common under the age of 65. It presents with changes in behaviour and loss of insight more commonly than memory problems. Patients are disinhibited, apathetic and can be compulsive overeaters. They may have a normal MMSE, visuospatial and language function but they fail on tasks of verbal fluency and executive function.

Alzheimer's disease is the most common cause of dementia and presents with memory problems, difficulties with activities of daily living and behavioural changes.

Characteristic features of dementia with Lewy bodies (DLB) are fluctuation of awareness from day-to-day, signs of parkinsonism and visual hallucinations or delusions occur frequently.

Parkinson's disease dementia has a similar pathological process to DLB and both are associated with progressive cognitive decline and parkinsonism. The dementia associated with Parkinson's disease occurs more than a year after the development of the motor symptoms of the disease.

Vascular dementia classically presents with periods of sudden decline and then stability with a stepwise progression. Patients have a history of stroke, hypertension or ischaemic heart disease.



A 59-year-old man presented to his GP with wasting and weakness of the muscles in his left hand.

He had noticed severely impaired hand grip and had noticed problems with writing. He had also developed a tingling sensation over the palm of his hand extending up the forearm. Otherwise he felt well in himself and had not noticed any weakness or wasting of the right hand or lower limb. His weight was stable and he denied any neck problems or swallowing difficulties.

He had a past medical history of rheumatoid arthritis and hypertension treated with bendroflumethiazide and ibuprofen. He was a smoker of 10 cigarettes per day and consumed 4 units of alcohol per week.

On examination he appeared alert and orientated. Fundoscopy and cranial nerve examination were all normal and neck movements were full. On examination of the upper limb, there was significant wasting over the left thenar eminence and fasciculations with a small burn over the left thumb. No other fasciculations could be detected in the proximal limb or other hand. Tone appeared normal and reflexes were intact. There was weakness of thumb abduction and opposition, with loss of pinprick and light touch sensation over the thumb, index and middle finger. On examination of the lower limb, no abnormalities could be found.

Nerve conduction studies showed:

- Absent sensory action potential in median palmar branches.
- Denervation of the abductor pollicis brevis.
- Normal sensory and motor conduction studies in the right upper limb and lower limbs.

Given the above history and clinical findings, what is the most likely diagnosis?

(Please select 1 option)

○ Ulnar nerve palsy

○ Thoracic inlet syndrome

○ Syringomyelia

○ Median nerve palsy

○ Motor neurone disease

● Median nerve palsy Correct

○ Motor neurone disease

Key Learning Points

Neurology

- Median nerve palsy affects the muscles of the thenar eminence, the abductor pollicis (C7, C8), flexor pollicis brevis, opponens pollicis, and the lateral two lumbricals

Explanation

This is the typical appearance of carpal tunnel syndrome (CTS) arising as a result of pressure on the median nerve in the carpal tunnel.

The median nerve supplies the muscles of the thenar eminence (which is severely wasted) the abductor pollicis (C7, C8), flexor pollicis brevis and opponens pollicis, and the lateral two lumbricals. The nerve conduction studies confirm marked denervation and absent sensory potentials within the median nerve territory.

There are no features to suggest a diagnosis of motor neurone disease, which presents with a combination of both upper and lower motor neurone abnormalities in absence of sensory disturbance.

Syringomyelia within the cervical cord would cause lower motor neurone signs at the level of the syrinx, with dissociated pain and temperature loss and upper motor neurone signs in the legs.

Thoracic inlet syndrome affecting C8, T1 of the brachial plexus would cause additional weakness of hand muscles.

An ulnar nerve palsy would cause weakness of small muscles of the hand with preserved thenar muscle function.

Next Question

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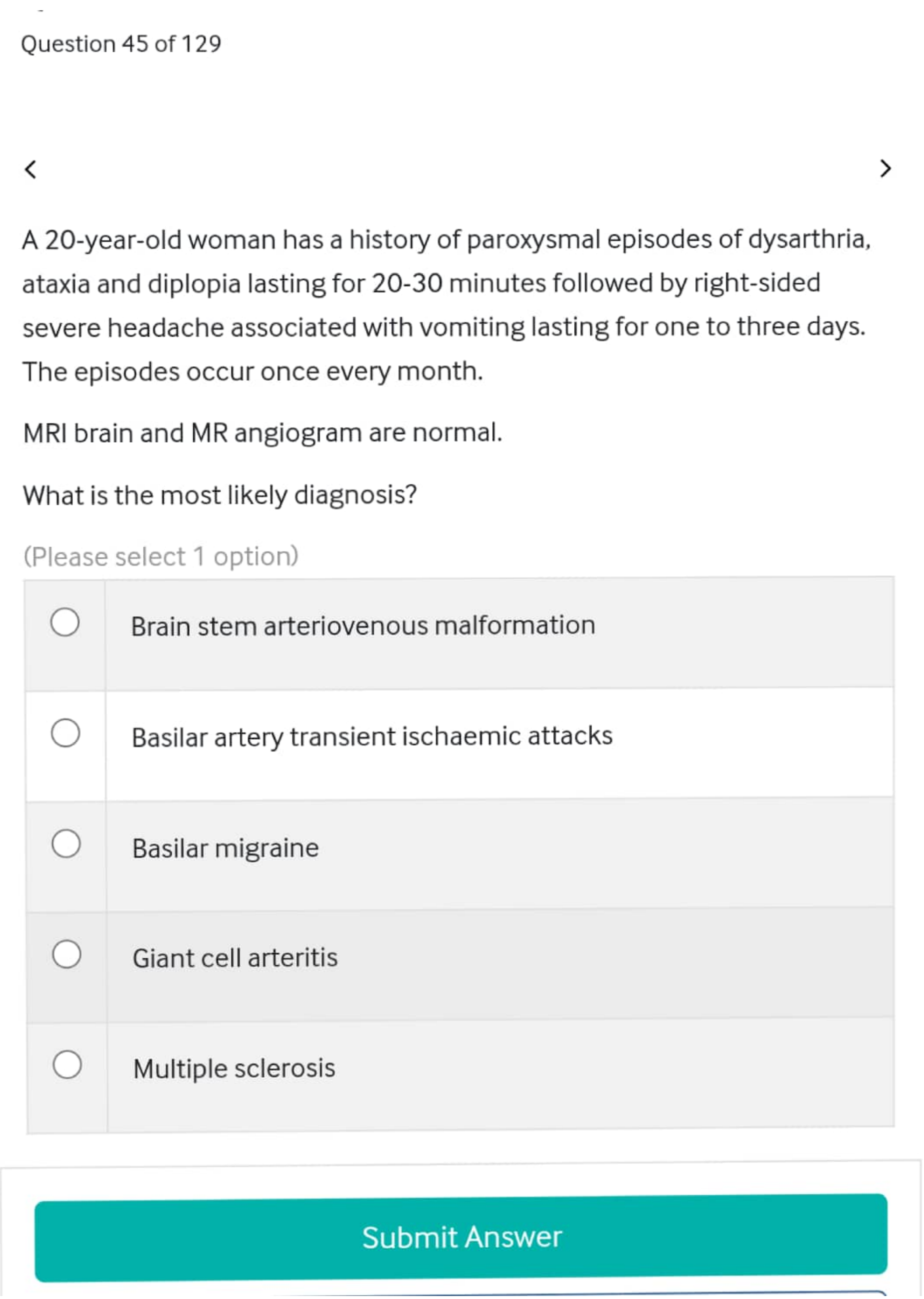


Question 45 of 129

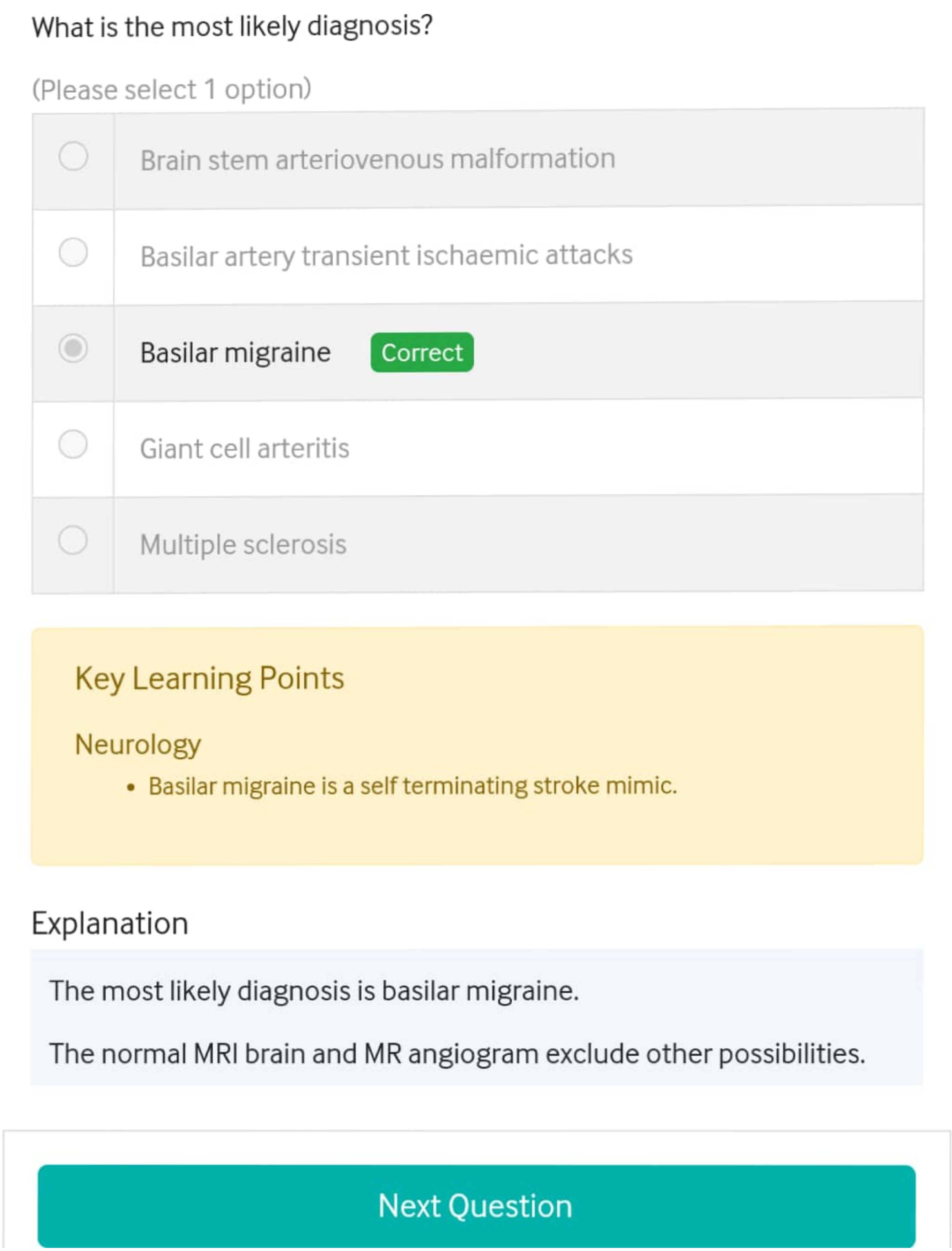
A 20-year-old woman has a history of paroxysmal episodes of dysarthria, ataxia and diplopia lasting for 20-30 minutes followed by right-sided severe headache associated with vomiting lasting for one to three days. The episodes occur once every month.

MRI brain and MR angiogram are normal.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Brain stem arteriovenous malformation
- ○ Basilar artery transient ischaemic attacks
- ○ Basilar migraine
- ○ Giant cell arteritis
- ○ Multiple sclerosis



What is the most likely diagnosis?

(Please select 1 option)

- ○ Brain stem arteriovenous malformation
- ○ Basilar artery transient ischaemic attacks
- ● Basilar migraine **Correct**
- ○ Giant cell arteritis
- ○ Multiple sclerosis

Key Learning Points

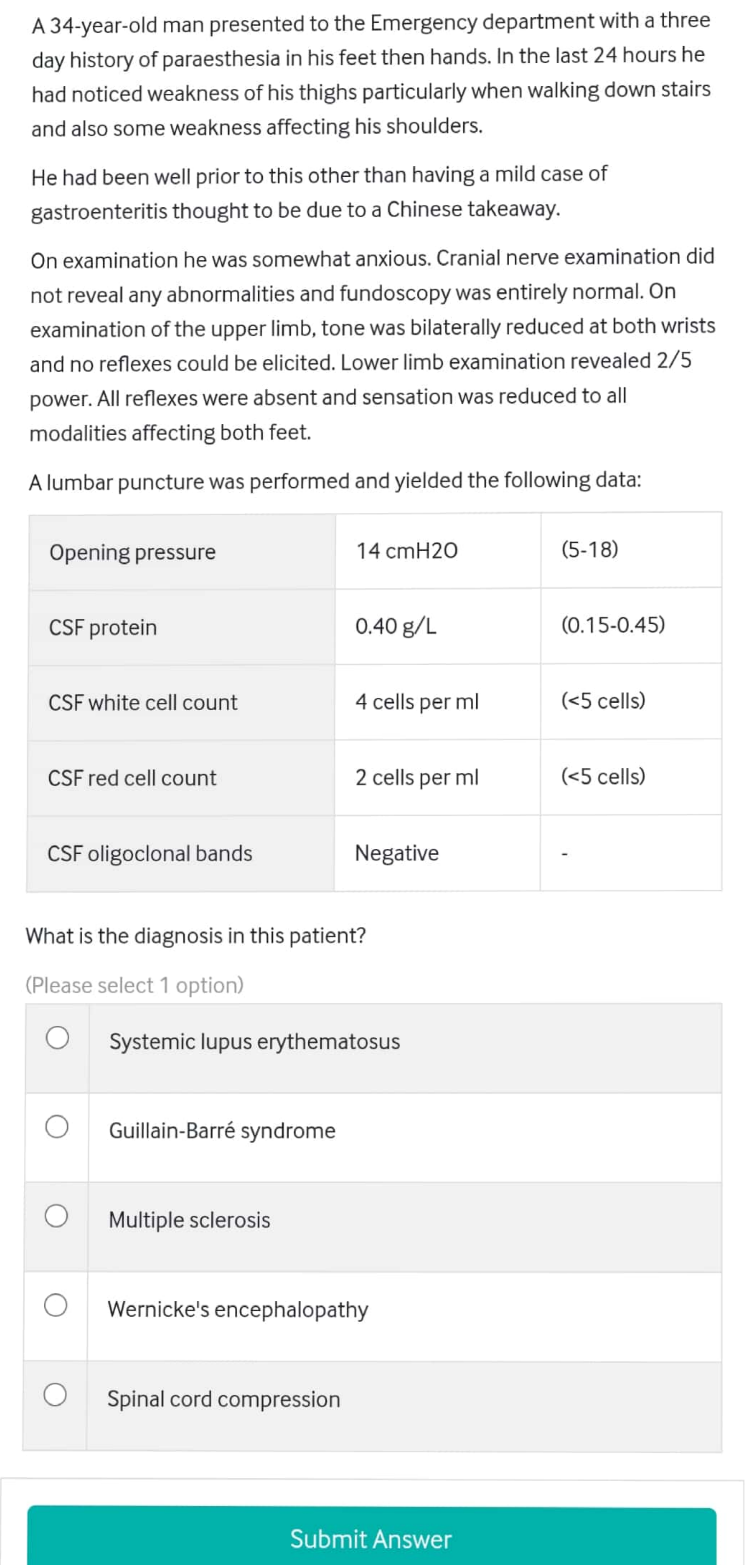
Neurology

- Basilar migraine is a self terminating stroke mimic.

Explanation

The most likely diagnosis is basilar migraine.

The normal MRI brain and MR angiogram exclude other possibilities.



A 34-year-old man presented to the Emergency department with a three day history of paraesthesia in his feet then hands. In the last 24 hours he had noticed weakness of his thighs particularly when walking down stairs and also some weakness affecting his shoulders.

He had been well prior to this other than having a mild case of gastroenteritis thought to be due to a Chinese takeaway.

On examination he was somewhat anxious. Cranial nerve examination did not reveal any abnormalities and fundoscopy was entirely normal. On examination of the upper limb, tone was bilaterally reduced at both wrists and no reflexes could be elicited. Lower limb examination revealed 2/5 power. All reflexes were absent and sensation was reduced to all modalities affecting both feet.

A lumbar puncture was performed and yielded the following data:

Opening pressure	14 cmH2O	(5-18)
CSF protein	0.40 g/L	(0.15-0.45)
CSF white cell count	4 cells per ml	(<5 cells)
CSF red cell count	2 cells per ml	(<5 cells)
CSF oligoclonal bands	Negative	-

What is the diagnosis in this patient?

(Please select 1 option)

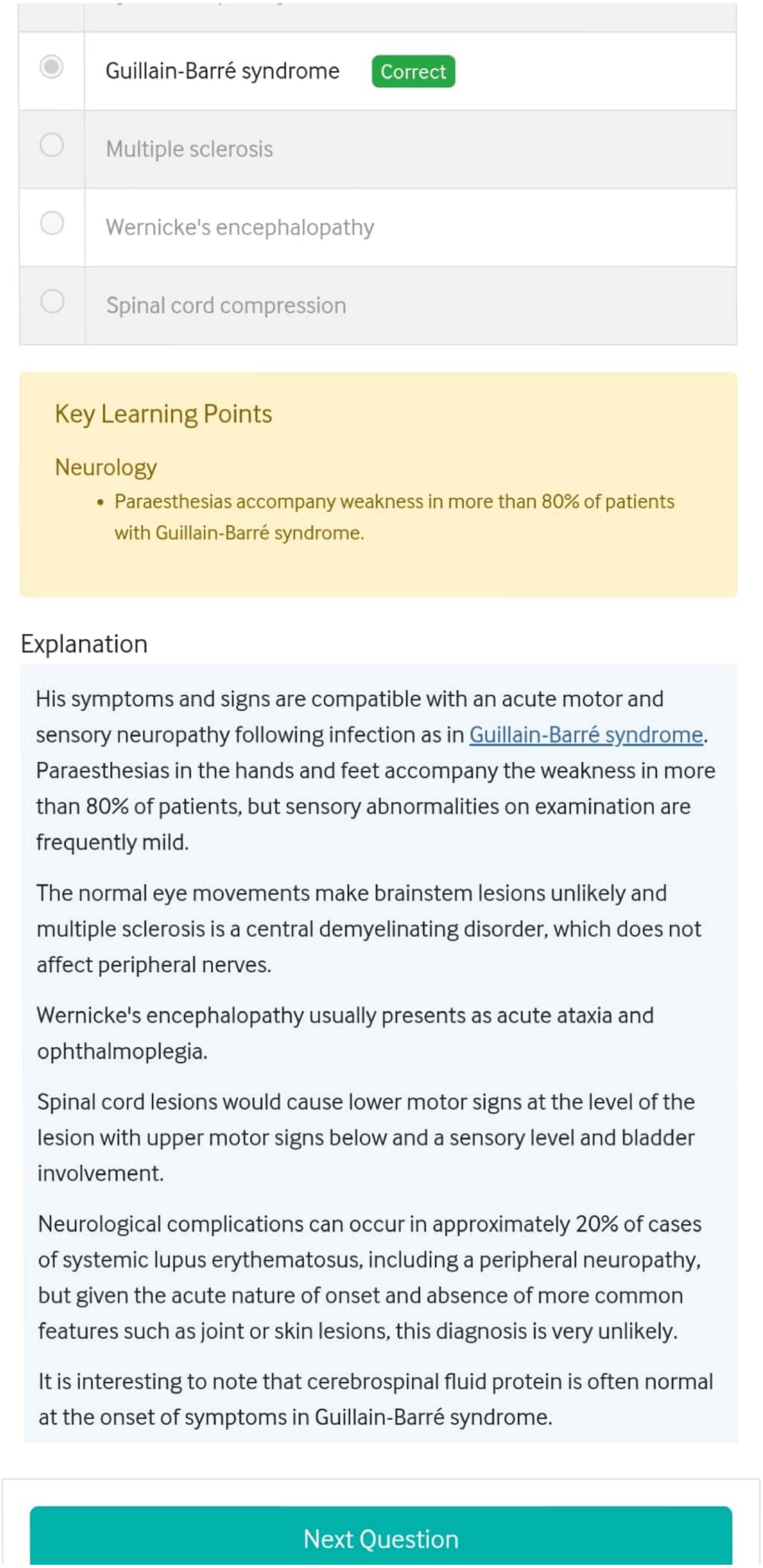
○ Systemic lupus erythematosus

○ Guillain-Barré syndrome

○ Multiple sclerosis

○ Wernicke's encephalopathy

○ Spinal cord compression



● Guillain-Barré syndrome **Correct**

○ Multiple sclerosis

○ Wernicke's encephalopathy

○ Spinal cord compression

Key Learning Points

Neurology

- Paraesthesias accompany weakness in more than 80% of patients with Guillain-Barré syndrome.

Explanation

His symptoms and signs are compatible with an acute motor and sensory neuropathy following infection as in Guillain-Barré syndrome. Paraesthesias in the hands and feet accompany the weakness in more than 80% of patients, but sensory abnormalities on examination are frequently mild.

The normal eye movements make brainstem lesions unlikely and multiple sclerosis is a central demyelinating disorder, which does not affect peripheral nerves.

Wernicke's encephalopathy usually presents as acute ataxia and ophthalmoplegia.

Spinal cord lesions would cause lower motor signs at the level of the lesion with upper motor signs below and a sensory level and bladder involvement.

Neurological complications can occur in approximately 20% of cases of systemic lupus erythematosus, including a peripheral neuropathy, but given the acute nature of onset and absence of more common features such as joint or skin lesions, this diagnosis is very unlikely.

It is interesting to note that cerebrospinal fluid protein is often normal at the onset of symptoms in Guillain-Barré syndrome.

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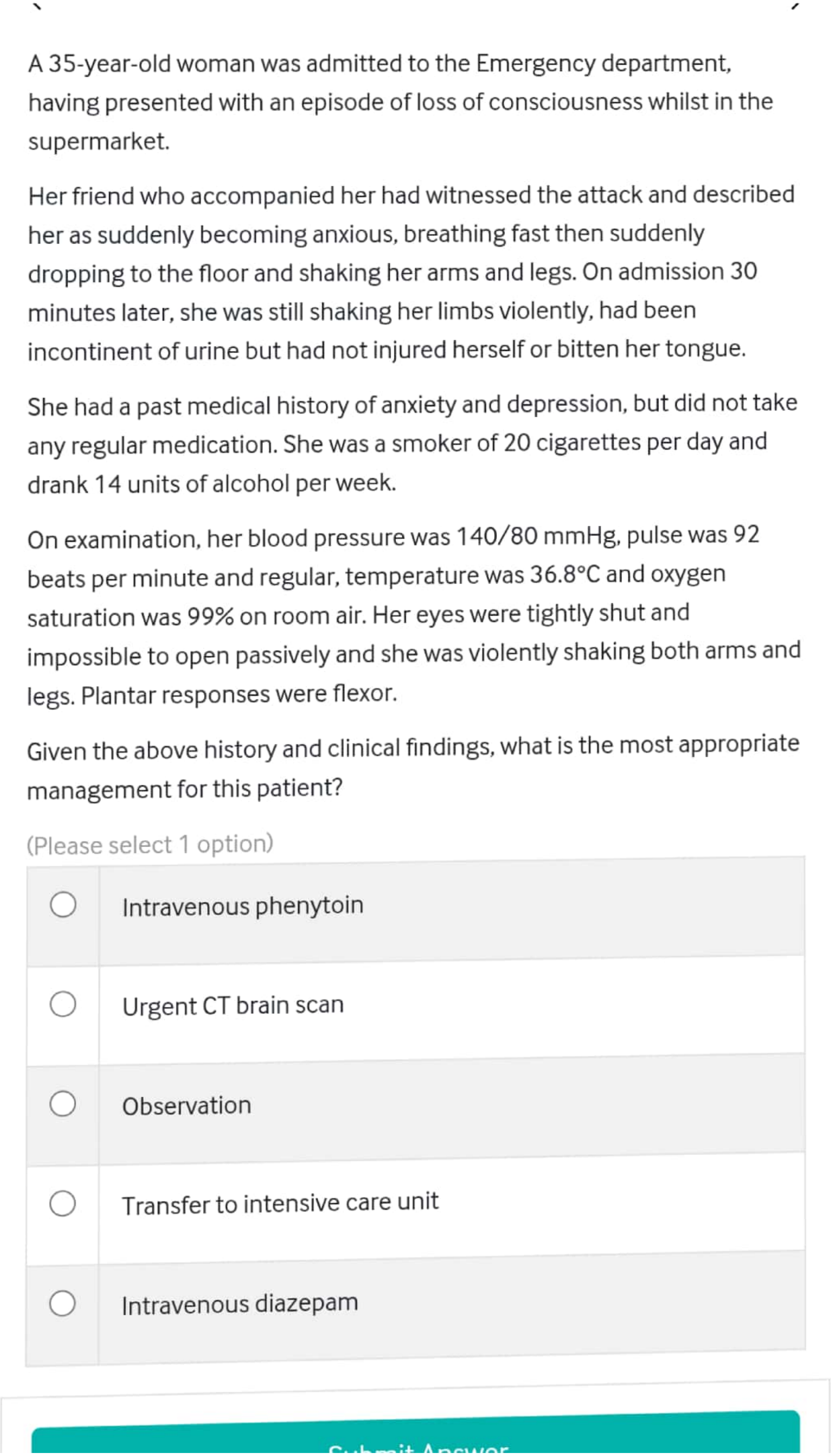

A 35-year-old woman was admitted to the Emergency department, having presented with an episode of loss of consciousness whilst in the supermarket.

Her friend who accompanied her had witnessed the attack and described her as suddenly becoming anxious, breathing fast then suddenly dropping to the floor and shaking her arms and legs. On admission 30 minutes later, she was still shaking her limbs violently, had been incontinent of urine but had not injured herself or bitten her tongue.

She had a past medical history of anxiety and depression, but did not take any regular medication. She was a smoker of 20 cigarettes per day and drank 14 units of alcohol per week.

On examination, her blood pressure was 140/80 mmHg, pulse was 92 beats per minute and regular, temperature was 36.8°C and oxygen saturation was 99% on room air. Her eyes were tightly shut and impossible to open passively and she was violently shaking both arms and legs. Plantar responses were flexor.

Given the above history and clinical findings, what is the most appropriate management for this patient?

(Please select 1 option)

○ Intravenous phenytoin

○ Urgent CT brain scan

○ Observation

○ Transfer to intensive care unit

○ Intravenous diazepam

○ Urgent CT brain scan

● Observation **Correct**

○ Transfer to intensive care unit

○ Intravenous diazepam

Key Learning Points

Neurology

- Violent shaking, resistance to passive eye opening and normal vital signs usually indicate a pseudoseizure.

Explanation

The clinical presentation is typical of pseudoseizure (non-epileptic attack).

Violent shaking, resistance to passive eye opening and normal vital signs are useful indicators of pseudoseizures.

Urinary incontinence can also occur in pseudoseizures but tongue biting is rare.

Simple observation is the appropriate management in this case.

Pseudoseizures are commonly misdiagnosed as true seizures and treated inappropriately with anti-epileptic drugs.

Next Question

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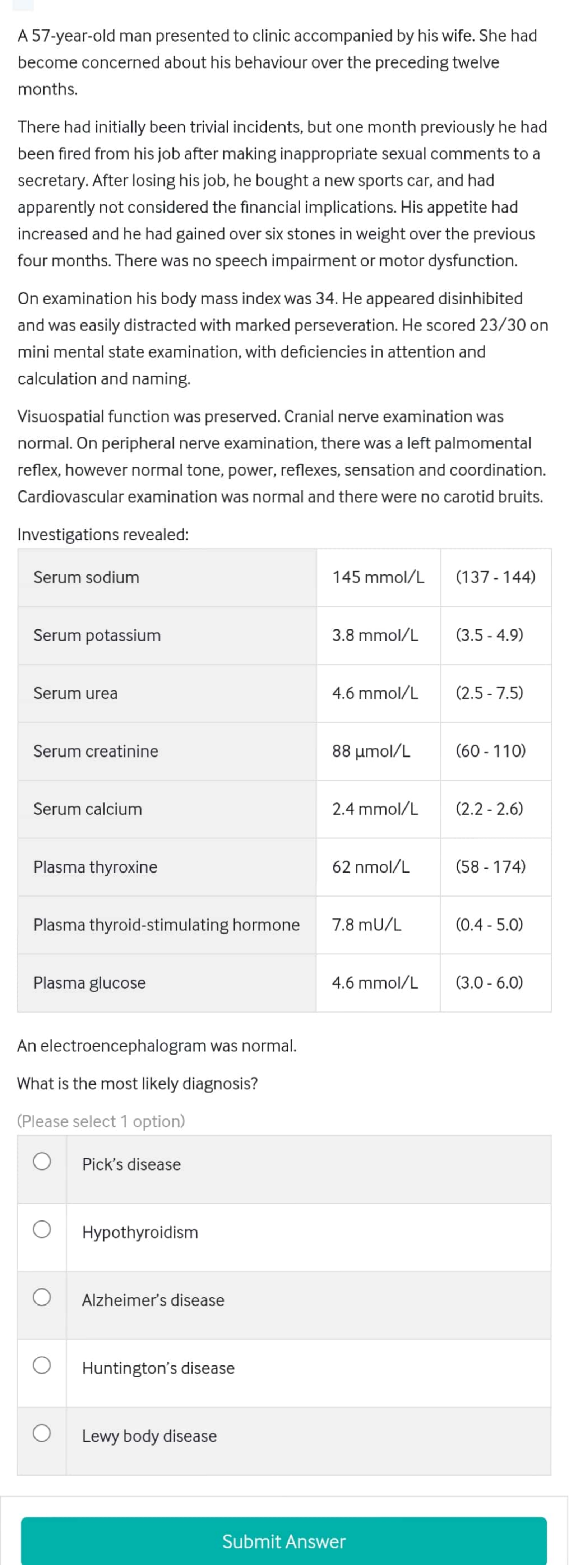


A 57-year-old man presented to clinic accompanied by his wife. She had become concerned about his behaviour over the preceding twelve months.

There had initially been trivial incidents, but one month previously he had been fired from his job after making inappropriate sexual comments to a secretary. After losing his job, he bought a new sports car, and had apparently not considered the financial implications. His appetite had increased and he had gained over six stones in weight over the previous four months. There was no speech impairment or motor dysfunction.

On examination his body mass index was 34. He appeared disinhibited and was easily distracted with marked perseveration. He scored 23/30 on mini mental state examination, with deficiencies in attention and calculation and naming.

Visuospatial function was preserved. Cranial nerve examination was normal. On peripheral nerve examination, there was a left palmomental reflex, however normal tone, power, reflexes, sensation and coordination. Cardiovascular examination was normal and there were no carotid bruits.

Investigations revealed:

Serum sodium	145 mmol/L	(137 - 144)
Serum potassium	3.8 mmol/L	(3.5 - 4.9)
Serum urea	4.6 mmol/L	(2.5 - 7.5)
Serum creatinine	88 μmol/L	(60 - 110)
Serum calcium	2.4 mmol/L	(2.2 - 2.6)
Plasma thyroxine	62 nmol/L	(58 - 174)
Plasma thyroid-stimulating hormone	7.8 mU/L	(0.4 - 5.0)
Plasma glucose	4.6 mmol/L	(3.0 - 6.0)

An electroencephalogram was normal.

What is the most likely diagnosis?

(Please select 1 option)

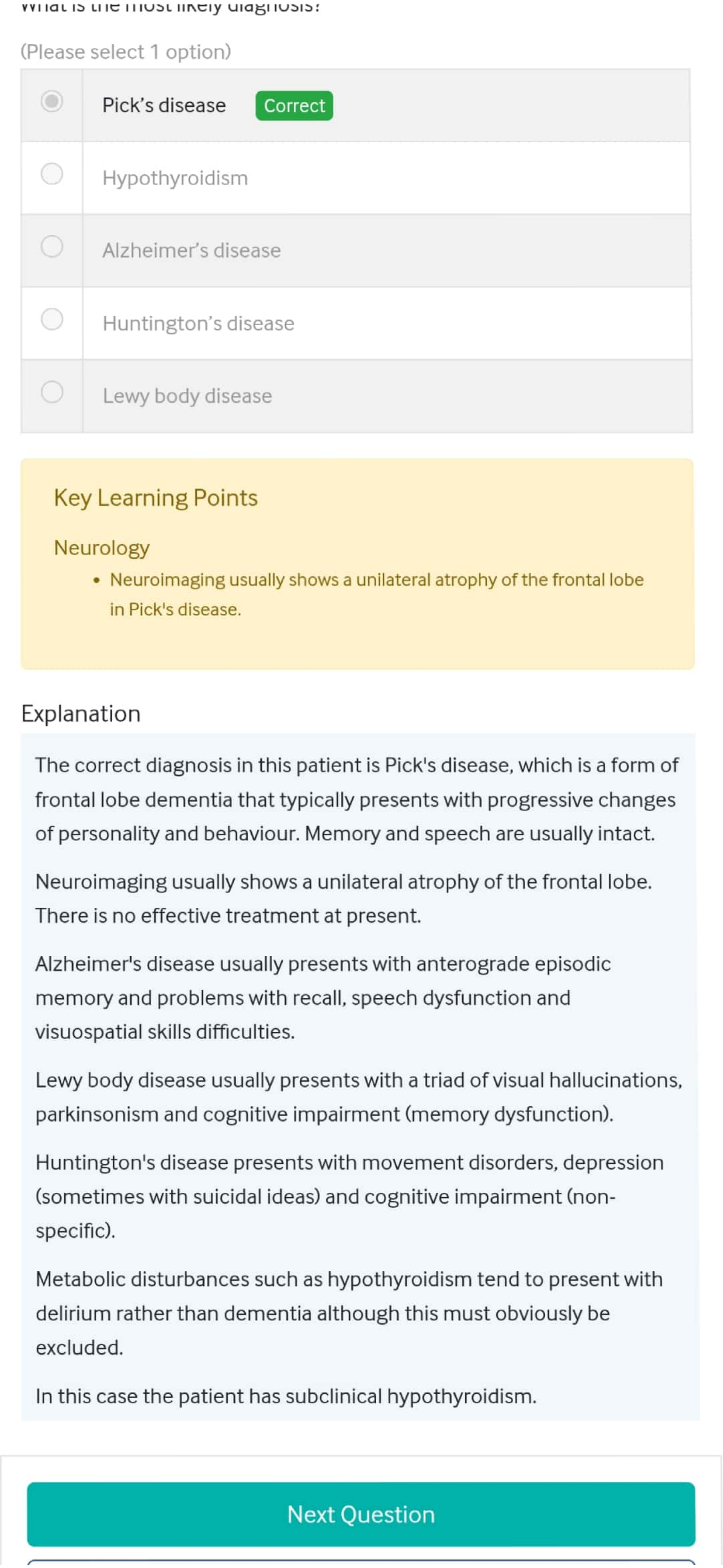
○ Pick's disease

○ Hypothyroidism

○ Alzheimer's disease

○ Huntington's disease

○ Lewy body disease



What is the most likely diagnosis?

(Please select 1 option)

- ● Pick's disease
- ○ Hypothyroidism
- ○ Alzheimer's disease
- ○ Huntington's disease
- ○ Lewy body disease

Key Learning Points

Neurology

- Neuroimaging usually shows a unilateral atrophy of the frontal lobe in Pick's disease.

Explanation

The correct diagnosis in this patient is Pick's disease, which is a form of frontal lobe dementia that typically presents with progressive changes of personality and behaviour. Memory and speech are usually intact.

Neuroimaging usually shows a unilateral atrophy of the frontal lobe. There is no effective treatment at present.

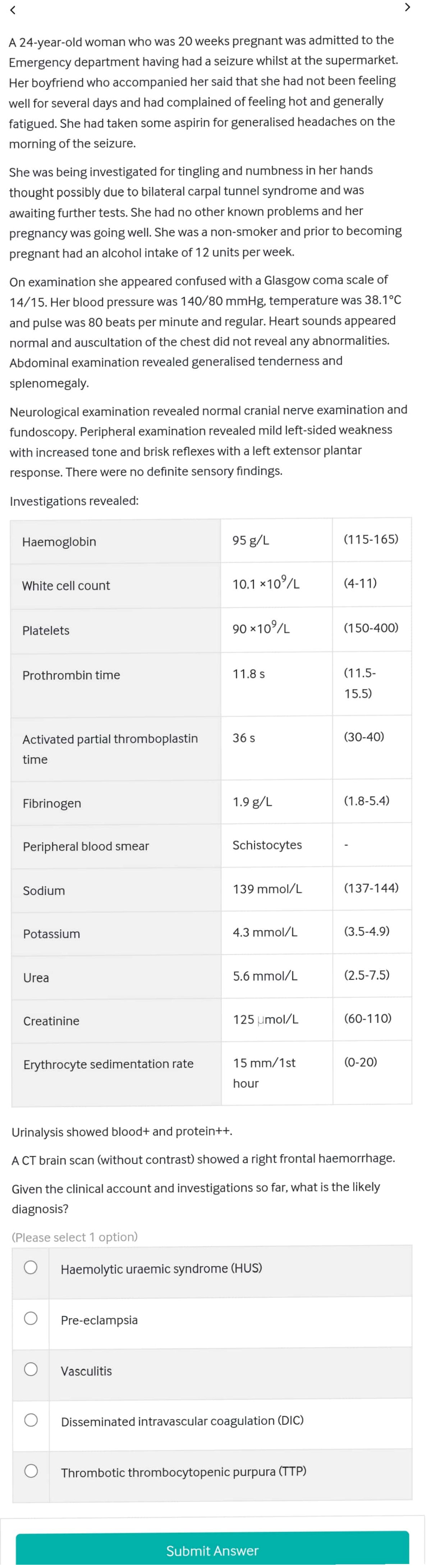
Alzheimer's disease usually presents with anterograde episodic memory and problems with recall, speech dysfunction and visuospatial skills difficulties.

Lewy body disease usually presents with a triad of visual hallucinations, parkinsonism and cognitive impairment (memory dysfunction).

Huntington's disease presents with movement disorders, depression (sometimes with suicidal ideas) and cognitive impairment (non-specific).

Metabolic disturbances such as hypothyroidism tend to present with delirium rather than dementia although this must obviously be excluded.

In this case the patient has subclinical hypothyroidism.



A 24-year-old woman who was 20 weeks pregnant was admitted to the Emergency department having had a seizure whilst at the supermarket. Her boyfriend who accompanied her said that she had not been feeling well for several days and had complained of feeling hot and generally fatigued. She had taken some aspirin for generalised headaches on the morning of the seizure.

She was being investigated for tingling and numbness in her hands thought possibly due to bilateral carpal tunnel syndrome and was awaiting further tests. She had no other known problems and her pregnancy was going well. She was a non-smoker and prior to becoming pregnant had an alcohol intake of 12 units per week.

On examination she appeared confused with a Glasgow coma scale of 14/15. Her blood pressure was 140/80 mmHg, temperature was 38.1°C and pulse was 80 beats per minute and regular. Heart sounds appeared normal and auscultation of the chest did not reveal any abnormalities. Abdominal examination revealed generalised tenderness and splenomegaly.

Neurological examination revealed normal cranial nerve examination and fundoscopy. Peripheral examination revealed mild left-sided weakness with increased tone and brisk reflexes with a left extensor plantar response. There were no definite sensory findings.

Investigations revealed:

Haemoglobin	95 g/L	(115-165)
White cell count	10.1 $\times 10^9$/L	(4-11)
Platelets	90 $\times 10^9$/L	(150-400)
Prothrombin time	11.8 s	(11.5-15.5)
Activated partial thromboplastin time	36 s	(30-40)
Fibrinogen	1.9 g/L	(1.8-5.4)
Peripheral blood smear	Schistocytes	-
Sodium	139 mmol/L	(137-144)
Potassium	4.3 mmol/L	(3.5-4.9)
Urea	5.6 mmol/L	(2.5-7.5)
Creatinine	125 μmol/L	(60-110)
Erythrocyte sedimentation rate	15 mm/1st hour	(0-20)

Urinalysis showed blood+ and protein++.

A CT brain scan (without contrast) showed a right frontal haemorrhage.

Given the clinical account and investigations so far, what is the likely diagnosis?

(Please select 1 option)

○ Haemolytic uraemic syndrome (HUS)

○ Pre-eclampsia

○ Vasculitis

○ Disseminated intravascular coagulation (DIC)

○ Thrombotic thrombocytopenic purpura (TTP)

○ Disseminated intravascular coagulation (DIC)

● Thrombotic thrombocytopenic purpura (TTP) Correct

Key Learning Points

Neurology

- TTP is treated with daily plasma exchange and high dose methylprednisolone for 3 days.

Explanation

This patient has presented with a seizure, right frontal haemorrhage and headaches.

She has a haemolytic anaemia, thrombocytopenia, and evidence of renal dysfunction. Her coagulation tests however are normal.

This patient has thrombotic thrombocytopenic purpura (TTP), a syndrome characterised by:

- Microangiopathic haemolytic anaemia
- Neurological abnormalities
- Fever, and
- Renal dysfunction.

Patients with TTP usually present with:

- Nonspecific complaints of fever, and
- Fatigue.

However, it can present with:

- Seizures
- Focal neurological deficit
- Paraesthesia
- Heart failure
- Arrhythmias, and
- Abdominal pain.

TTP is associated with pregnancy, HIV-1 infection and carcinomas.

Intravenous (IV) plasma exchange is the present standard of treatment for TTP and has improved the survival rate from approximately 3% prior to the 1960s to 82%.

HUS is closely related to TTP but is more common in children normally with a history of gastrointestinal infection.

DIC is a disorder of coagulation and fibrinogen/fibrin would be reduced.

Vasculitis can present in a similar way to TTP, but given a normal erythrocyte sedimentation rate is less likely.

Pre-eclampsia is less likely in the presence of splenomegaly and absence of significantly elevated systolic blood pressure.

Question 50 of 129

<   >

A 60-year-old woman presented with a three month history of unsteadiness and fatigue. In her past medical history she was diagnosed with type 2 diabetes mellitus four years ago, and one year ago had been diagnosed with hypothyroidism.

She was taking metformin 500 mg tds and thyroxine 100 µg daily. Her most recent thyroid function tests were normal.

On examination she had spastic paraparesis, absent knee and ankle reflexes, extensor plantar responses, absent joint position sense up to knees with normal pain sensation.

What is the most likely diagnosis?

(Please select 1 option)

○ Spinal meningioma

○ Syringomyelia

○ Multiple sclerosis

○ Metastatic spinal disease

○ Subacute combined cord degeneration

Submit Answer

○ Multiple sclerosis

○ Metastatic spinal disease

● Subacute combined cord degeneration **Correct**

Key Learning Points

Neurology

- Subacute combined cord degeneration is due to vitamin B12 deficiency and is characterised by absent reflexes and extensor plantar response.

Explanation

Subacute combined cord degeneration is due to vitamin B_{12} deficiency.

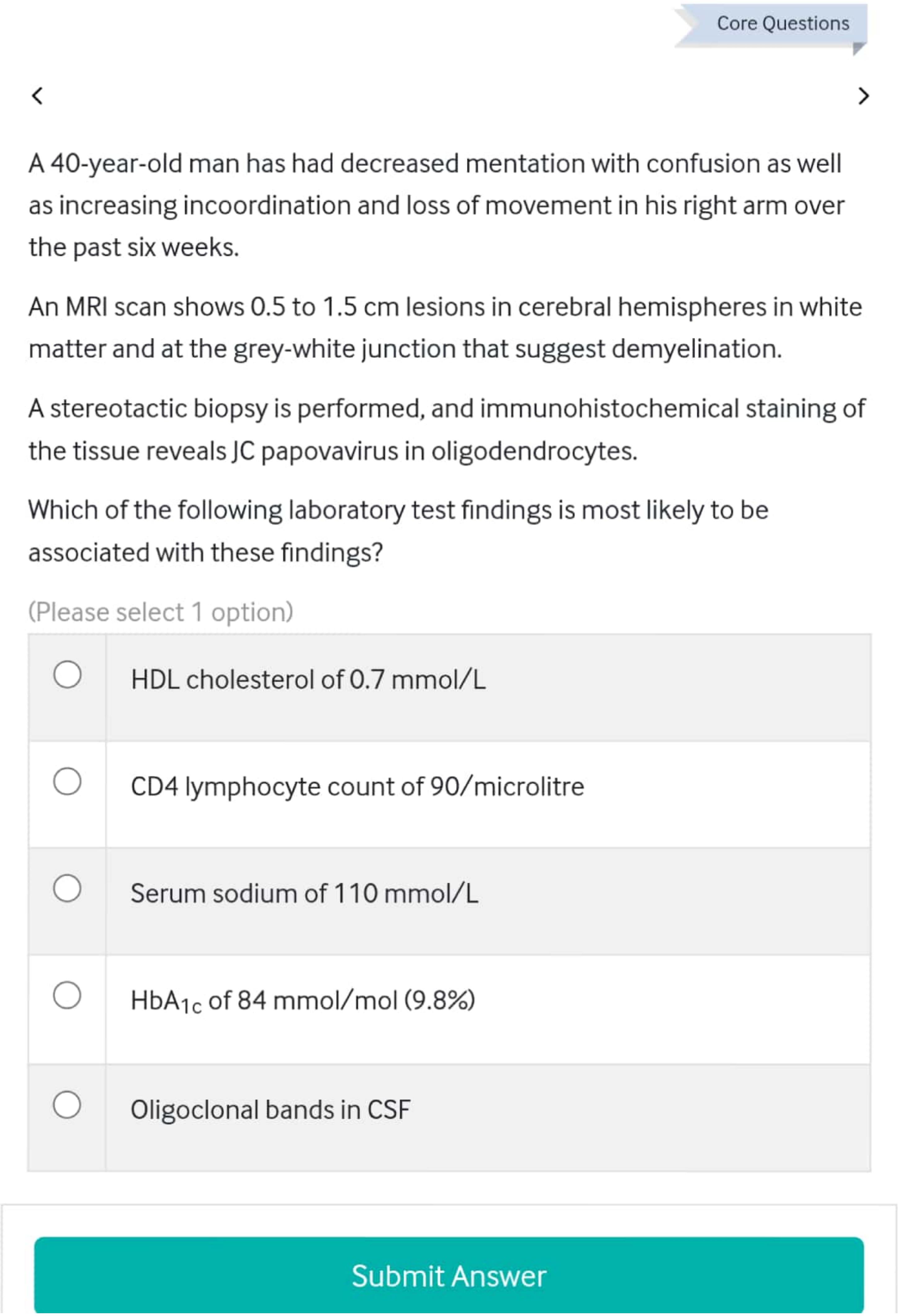
Pernicious anaemia is the commonest cause and is usually associated with other autoimmune diseases (for example, hypothyroidism and diabetes).

Prolonged metformin treatment has also been associated with B12 deficiency (in up to 10% of patients in a large four year clinical trial), therefore B12 levels should be screened every few years in patients taking long term metformin.

The combination of absent reflexes and extensor plantar response is typical and is due to co-existing peripheral neuropathy.

Dorsal column involvement with relative sparing of spinothalamic pathways is also typical.

Next Question



A 40-year-old man has had decreased mentation with confusion as well as increasing incoordination and loss of movement in his right arm over the past six weeks.

An MRI scan shows 0.5 to 1.5 cm lesions in cerebral hemispheres in white matter and at the grey-white junction that suggest demyelination.

A stereotactic biopsy is performed, and immunohistochemical staining of the tissue reveals JC papovavirus in oligodendrocytes.

Which of the following laboratory test findings is most likely to be associated with these findings?

(Please select 1 option)

○ HDL cholesterol of 0.7 mmol/L

○ CD4 lymphocyte count of 90/microlitre

○ Serum sodium of 110 mmol/L

○ HbA_{1c} of 84 mmol/mol (9.8%)

○ Oligoclonal bands in CSF

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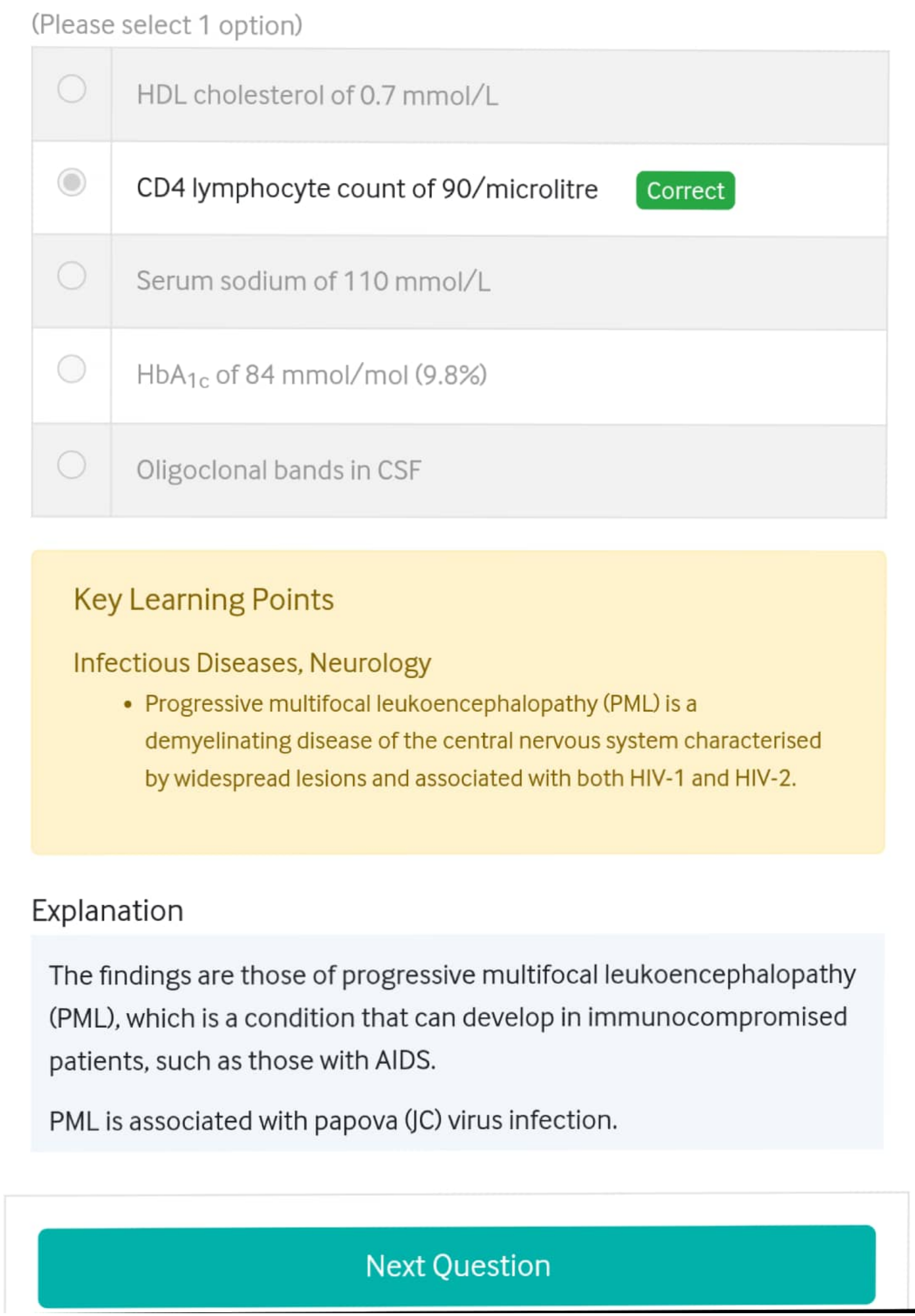


(Please select 1 option)

○ HDL cholesterol of 0.7 mmol/L

● CD4 lymphocyte count of 90/microlitre Correct

○ Serum sodium of 110 mmol/L

○ HbA$_{1c}$ of 84 mmol/mol (9.8%)

○ Oligoclonal bands in CSF

Key Learning Points

Infectious Diseases, Neurology

- Progressive multifocal leukoencephalopathy (PML) is a demyelinating disease of the central nervous system characterised by widespread lesions and associated with both HIV-1 and HIV-2.

Explanation

The findings are those of progressive multifocal leukoencephalopathy (PML), which is a condition that can develop in immunocompromised patients, such as those with AIDS.

PML is associated with papova (JC) virus infection.

A 65-year-old Asian lady was referred to the emergency department presenting with a history of nausea, vomiting and headache which she had had for several days. She did not speak English and unfortunately there was no-one to translate her history.

She had an accompanying GP letter which revealed she had registered with the practice recently, having emigrated from India with her son. She was a known type 2 diabetic and also suffered from atrial fibrillation. Her regular medications included metformin, digoxin and warfarin.

On examination she appeared apathetic and drowsy with a Glasgow coma scale of 14/15. Her blood pressure was 145/75 mmHg, pulse was 76/min and irregularly irregular and temperature was 36.2°C.

There were several bruises to her arms and legs. Heart sounds revealed a harsh pansystolic murmur, heard best at the apex, which was displaced. Chest and gastrointestinal examinations were entirely normal.

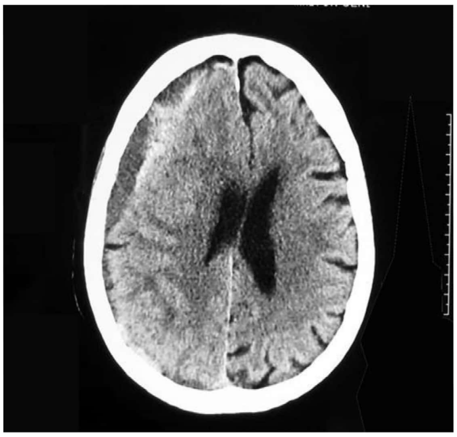
Neurological examination was difficult given the language barrier; however fundoscopy revealed some nasal blurring in both eyes, impaired upward gaze and intact pupillary responses. There was no evidence of any facial asymmetry. Peripheral nervous system examination revealed some increased tone affecting the left arm and leg with minimal weakness, but pathologically brisk reflexes and a left extensor plantar response. Sensory examination was not assessed.

Investigations showed:

Haemoglobin	102 g/L	(115-165)
White cell count	11 $\times 10^9$/L	(4-11)
Platelets	357 $\times 10^9$/L	(150-400)
C reactive protein	14 mg/L	(<10)
Activated partial thromboplastin time	47 seconds	(30-40)
INR	3.0	
Glucose	5.2 mmol/L	(3.0-6.0)

The chest x ray was normal.

A non-enhanced CT brain scan is shown below:



A lumbar puncture was performed following interpretation of the CT imaging:

CSF Opening pressure	18 cmH_2O	(4-18)
CSF white cell count	7 cells per ml	(<5)
	90% lymphocytes	(60-70)
CSF red cell count	100 cells per ml	-
CSF protein	0.20 g/L	(0.15-0.45)
CSF glucose	3.7 mmol/L	(3.3-4.4)
Xanthochromia	Present	-

Given the above account and investigations so far, what is the most likely diagnosis in this patient?

(Please select 1 option)

○ Cerebral abscess

○ Subarachnoid haemorrhage

○ Tuberculoma

○ Primary cerebral lymphoma

○ Chronic subdural haematoma

○ Primary cerebral lymphoma

● Chronic subdural haematoma **Correct**

Key Learning Points

Neurology, Radiology

- Chronic subdural haematoma should be considered in patients with fluctuating neurology on blood thinners and minor trauma.

Explanation

This patient presents with a sparse history and clinical information. She has a fairly recent history of headache, nausea and vomiting in the presence of a left hemiparesis and restricted upward gaze. Her CT scan image shows a lesion within the right parietal area and midline shift consistent with a chronic subdural haematoma.

Clues to this diagnosis are the history of anticoagulation with multiple skin bruises, suggesting the possibility of over anticoagulation, in absence of pyrexia. The blood tests show she is anaemic and over anticoagulated with a normal white cell count.

The lumbar puncture, which should not have been attempted, shows cellular CSF with elevated pressure and xanthochromia with relatively low protein content, suggesting extension of blood into the subarachnoid space. These findings would also be consistent with chronic subdural haemorrhage.

Tuberculomas and tuberculosis meningitis should always be considered in ethnic minority groups. Tuberculomas behave like any primary malignant brain tumour and can present with focal neurology and may cause hydrocephalus if periventricular in origin.

Against this diagnosis would be a normal CXR, and relatively low CSF protein. Cerebral abscess is unlikely in view of normal inflammatory markers, white cell count and temperature.

Subarachnoid haemorrhage does not explain the brain lesion.

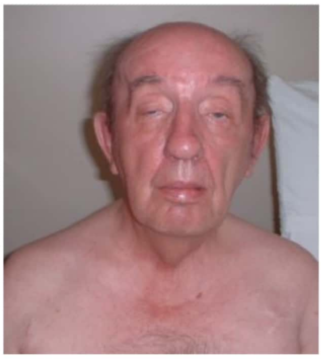
Primary CNS lymphoma present with seizures and focal signs, but the lesions are often confined to the corpus callosum.

Next Question

A 58-year-old man was seen in the neurology clinic having been referred with weakness of both hands and wrists.

In the preceding week he had been having several falls and felt he was dragging his feet whilst walking. He had recently been to see his GP because of weight loss and had been diagnosed with impaired glucose tolerance. Otherwise he suffered occasional palpitations, but had no known medical history and did not take any regular medication.

On examination the patient had a monotonous, nasal voice. His general appearance is shown in the photograph below:



Cranial nerve examination revealed bilateral ptosis, but the pupils and fundoscopy were intact. There was marked bilateral facial weakness and also some weakness of neck flexion and extension. Upper limb inspection revealed marked distal wasting of the hands and forearm musculature. There was reduced tone, supinator jerks bilaterally, weakness of finger flexion and extension and also wrist extension. There was some mild weakness of shoulder abduction bilaterally. Lower limb examination revealed wasting around the ankles with reduced ankle tone and reflexes and weakness of dorsiflexion and plantarflexion. Sensory examination was intact.

Investigations revealed:

Haemoglobin	152 g/L	(130-180)
Mean cell volume	93 fL	(80-96)
White cell count	5.1 $\times 10^9$/L	(4-11)
Platelets	221 $\times 10^9$/L	(150-400)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	6.6 mmol/L	(2.5-7.5)
Serum creatinine	61 μmol/L	(60-110)
Serum creatine kinase	170 U/L	(24-170)
Plasma lactate	1.2 mmol//L	(0.6-1.2)
Fasting plasma glucose	8.2 mmol/L	(3-6)
Plasma TSH	4.2 mU/L	(0.5-5.0)
Plasma T4	62 nmol/L	(58-174)
Plasma T3	1.99 nmol/L	(1.07-3.18)

His ECG showed a PR interval of 0.3 seconds.

What is the diagnosis?

(Please select 1 option)

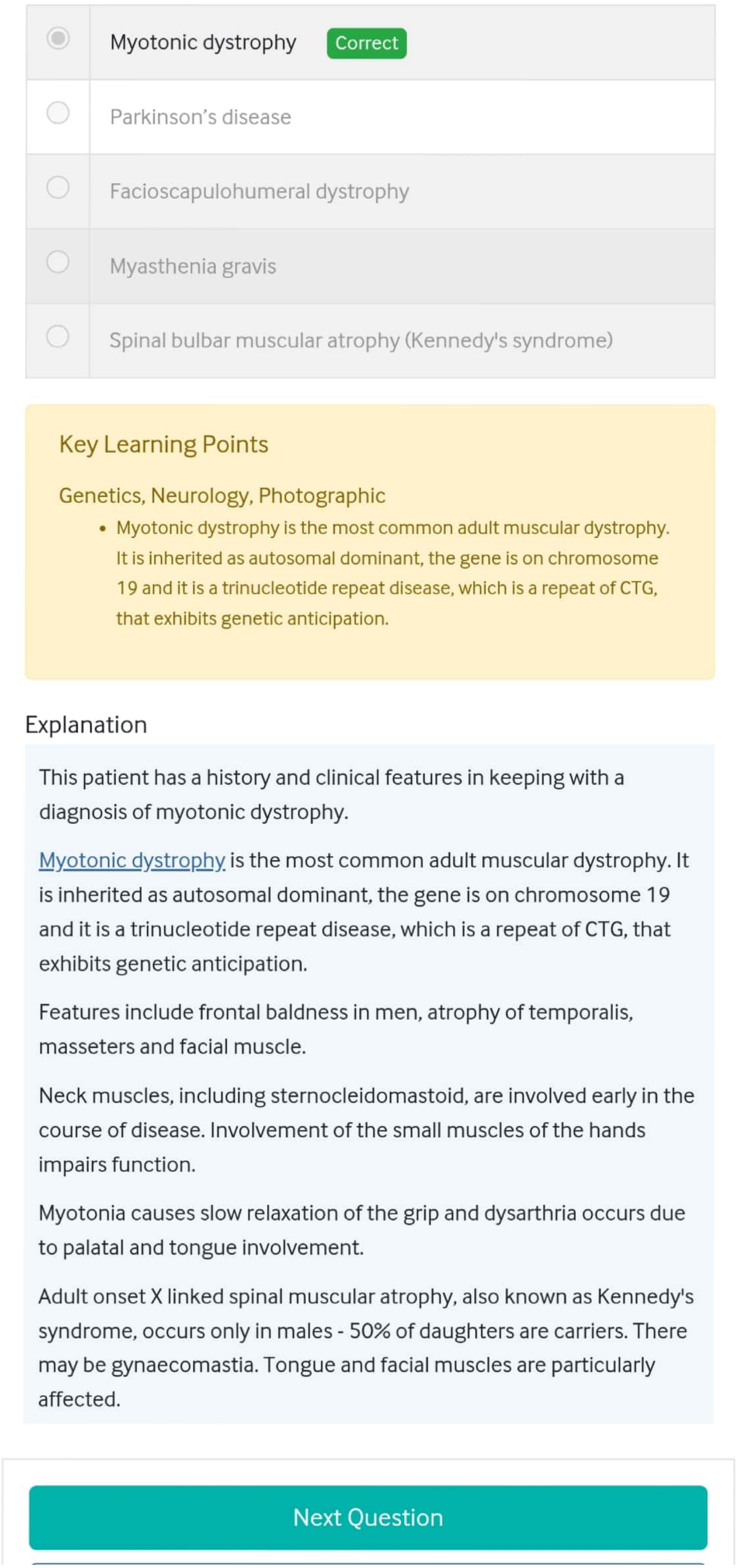
○ Myotonic dystrophy

○ Parkinson's disease

○ Facioscapulohumeral dystrophy

○ Myasthenia gravis

○ Spinal bulbar muscular atrophy (Kennedy's syndrome)



● Myotonic dystrophy

○ Parkinson’s disease

○ Facioscapulohumeral dystrophy

○ Myasthenia gravis

○ Spinal bulbar muscular atrophy (Kennedy's syndrome)

Key Learning Points

Genetics, Neurology, Photographic

- Myotonic dystrophy is the most common adult muscular dystrophy. It is inherited as autosomal dominant, the gene is on chromosome 19 and it is a trinucleotide repeat disease, which is a repeat of CTG, that exhibits genetic anticipation.

Explanation

This patient has a history and clinical features in keeping with a diagnosis of myotonic dystrophy.

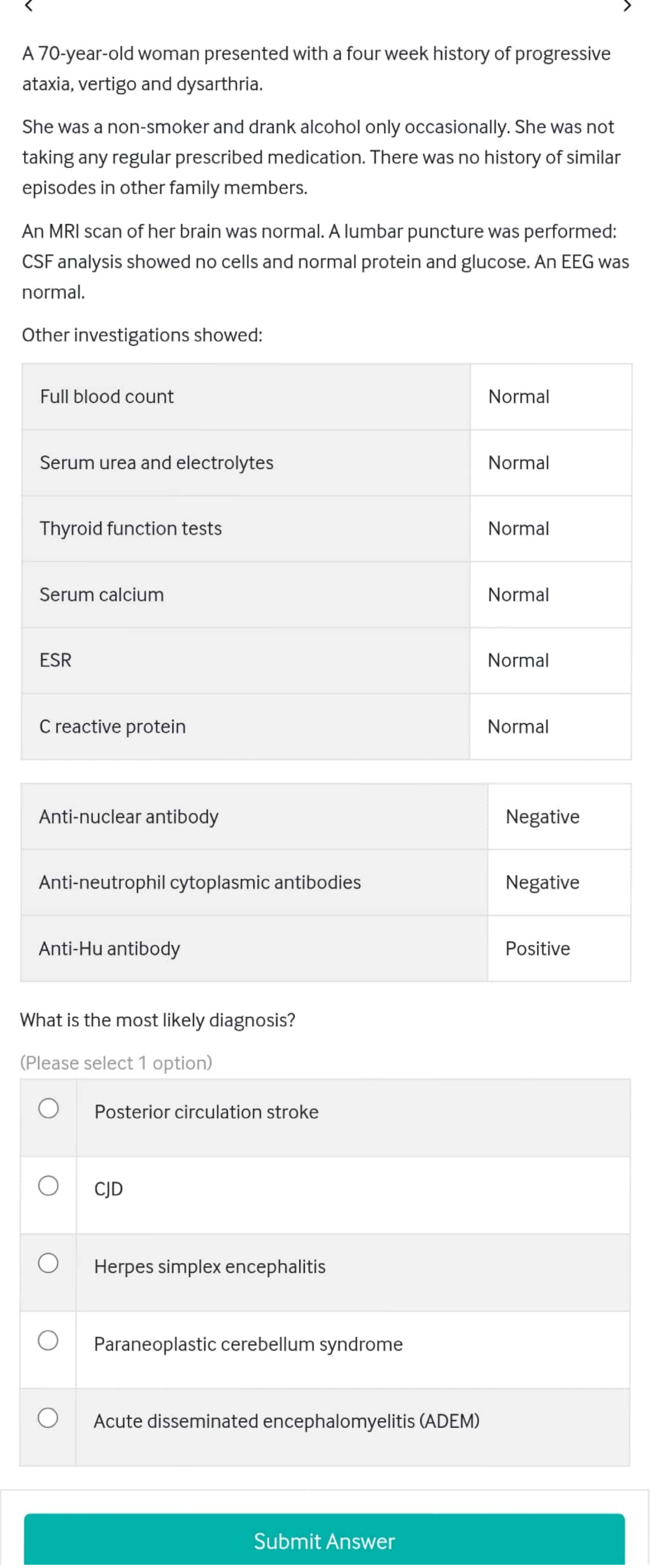
Myotonic dystrophy is the most common adult muscular dystrophy. It is inherited as autosomal dominant, the gene is on chromosome 19 and it is a trinucleotide repeat disease, which is a repeat of CTG, that exhibits genetic anticipation.

Features include frontal baldness in men, atrophy of temporalis, masseters and facial muscle.

Neck muscles, including sternocleidomastoid, are involved early in the course of disease. Involvement of the small muscles of the hands impairs function.

Myotonia causes slow relaxation of the grip and dysarthria occurs due to palatal and tongue involvement.

Adult onset X linked spinal muscular atrophy, also known as Kennedy's syndrome, occurs only in males - 50% of daughters are carriers. There may be gynaecomastia. Tongue and facial muscles are particularly affected.



A 70-year-old woman presented with a four week history of progressive ataxia, vertigo and dysarthria.

She was a non-smoker and drank alcohol only occasionally. She was not taking any regular prescribed medication. There was no history of similar episodes in other family members.

An MRI scan of her brain was normal. A lumbar puncture was performed: CSF analysis showed no cells and normal protein and glucose. An EEG was normal.

Other investigations showed:

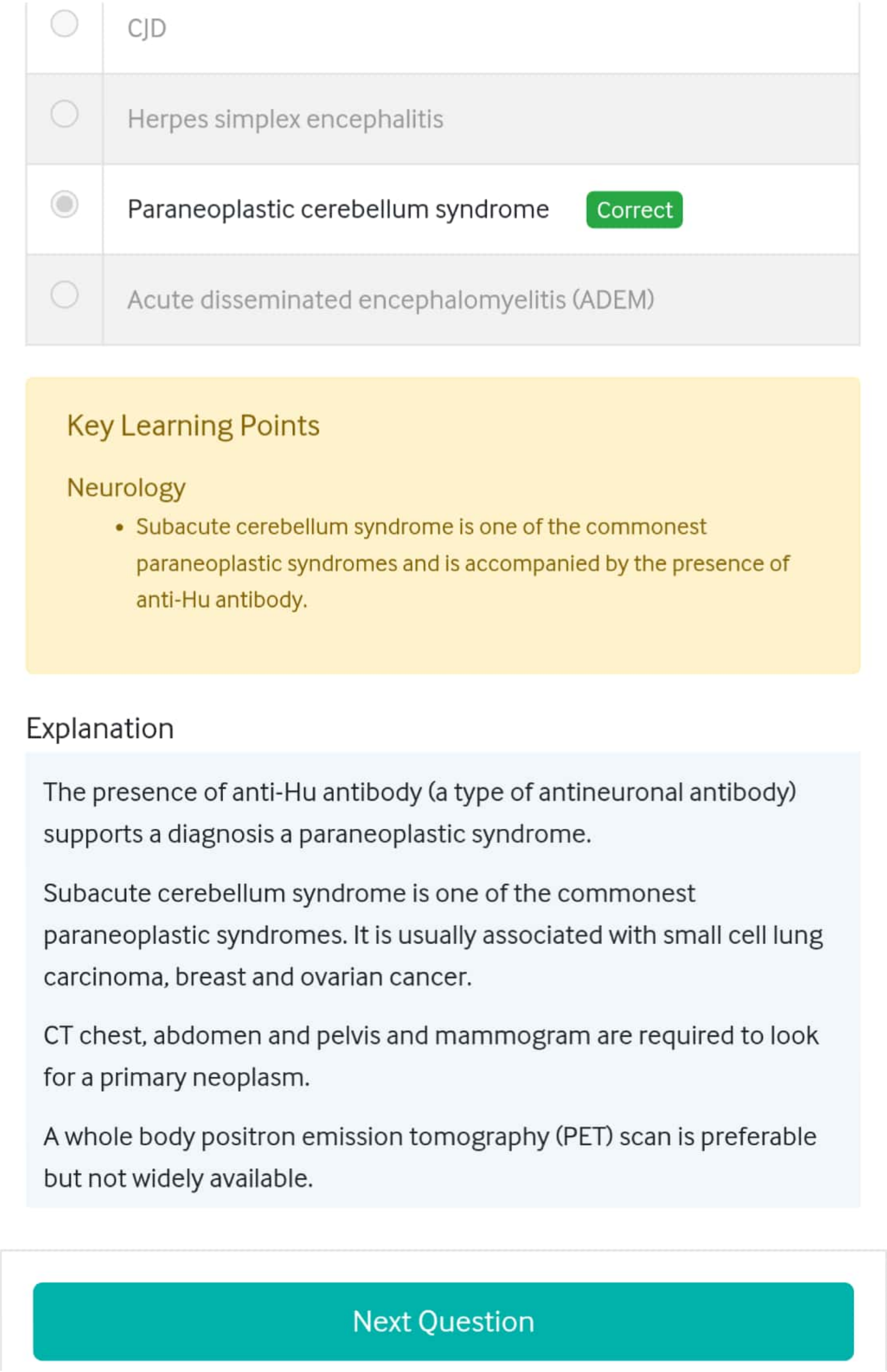
Full blood count	Normal
Serum urea and electrolytes	Normal
Thyroid function tests	Normal
Serum calcium	Normal
ESR	Normal
C reactive protein	Normal

Anti-nuclear antibody	Negative
Anti-neutrophil cytoplasmic antibodies	Negative
Anti-Hu antibody	Positive

What is the most likely diagnosis?

(Please select 1 option)

- ○ Posterior circulation stroke
- ○ CJD
- ○ Herpes simplex encephalitis
- ○ Paraneoplastic cerebellum syndrome
- ○ Acute disseminated encephalomyelitis (ADEM)



○ CJD

○ Herpes simplex encephalitis

● Paraneoplastic cerebellum syndrome **Correct**

○ Acute disseminated encephalomyelitis (ADEM)

Key Learning Points

Neurology

- Subacute cerebellum syndrome is one of the commonest paraneoplastic syndromes and is accompanied by the presence of anti-Hu antibody.

Explanation

The presence of anti-Hu antibody (a type of antineuronal antibody) supports a diagnosis a paraneoplastic syndrome.

Subacute cerebellum syndrome is one of the commonest paraneoplastic syndromes. It is usually associated with small cell lung carcinoma, breast and ovarian cancer.

CT chest, abdomen and pelvis and mammogram are required to look for a primary neoplasm.

A whole body positron emission tomography (PET) scan is preferable but not widely available.

A 45-year-old man presented with a six month history of agitation and delusions.

He had seen a psychiatrist who had made a diagnosis of paranoid schizophrenia and started depot haloperidol. His psychosis had improved considerably following this therapy and he had been well.

However, he was admitted by his GP following a three day history of confusion and increasing drowsiness.

On examination, he was agitated, with a temperature of 38.5°C and blood pressure of 190/110 mmHg. His cardiovascular and chest examination were normal and no enlarged liver, spleen or kidneys were noted. He had bilateral severe rigidity but no tremor.

Septic screen was normal.

What is the most likely diagnosis?

(Please select 1 option)

○ Herpes simplex encephalopathy

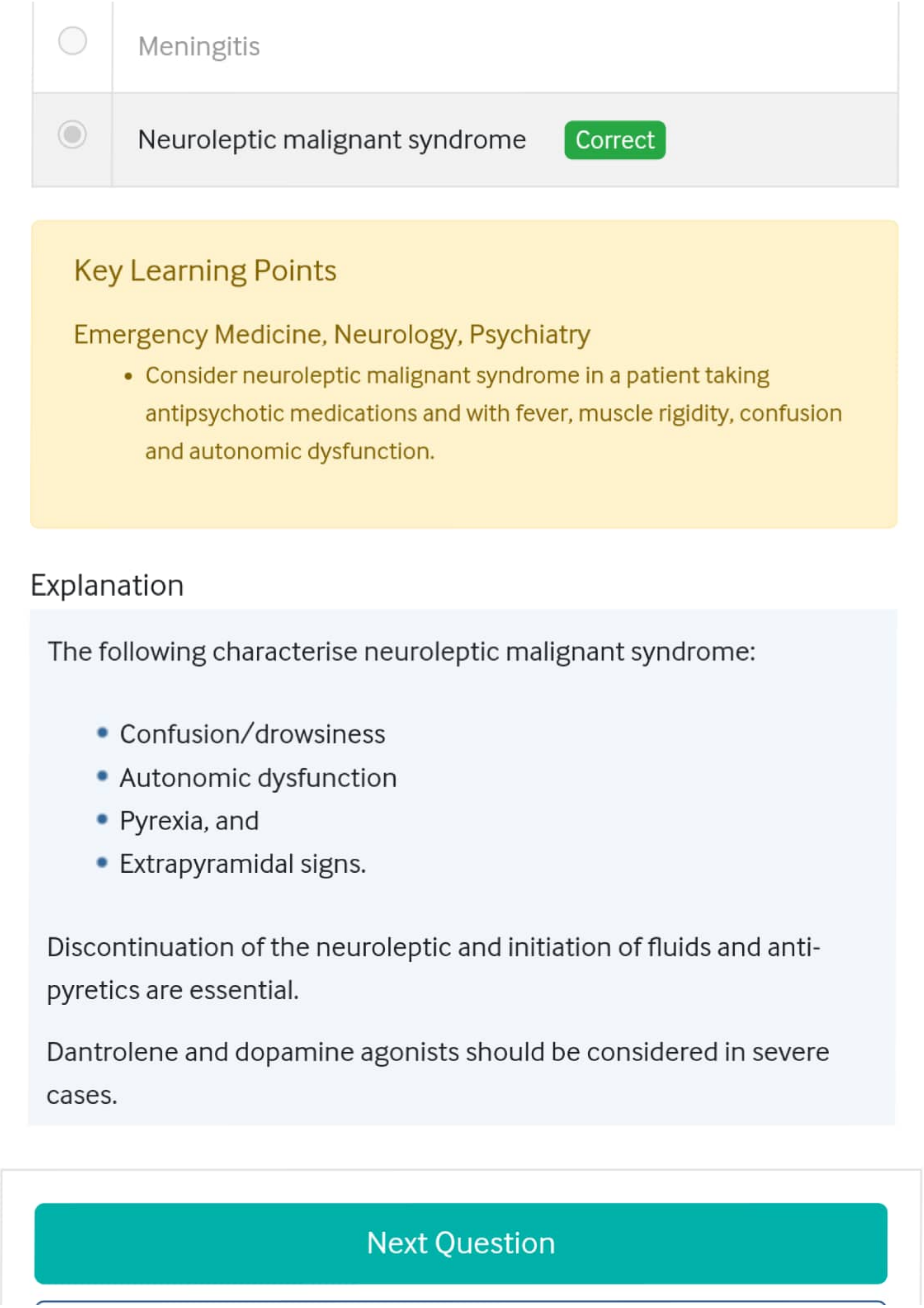
○ Non-convulsive status epilepticus

○ Hysteria

○ Meningitis

○ Neuroleptic malignant syndrome

Submit Answer



○ Meningitis

● Neuroleptic malignant syndrome

Key Learning Points

Emergency Medicine, Neurology, Psychiatry

- Consider neuroleptic malignant syndrome in a patient taking antipsychotic medications and with fever, muscle rigidity, confusion and autonomic dysfunction.

Explanation

The following characterise neuroleptic malignant syndrome:

- Confusion/drowsiness
- Autonomic dysfunction
- Pyrexia, and
- Extrapyramidal signs.

Discontinuation of the neuroleptic and initiation of fluids and anti-pyretics are essential.

Dantrolene and dopamine agonists should be considered in severe cases.

CamScanner الممسوحة ضوئيا بـ

A 46-year-old woman presents with sudden onset of severe occipital headache associated with nausea and vomiting.

Cerebrospinal fluid examination and a subsequent cerebral angiography confirm the diagnosis of an intracranial haemorrhage secondary to a bleed from a posterior communicating artery aneurysm. After consultation with the neurosurgeons, the patient is sent for surgical clipping of her aneurysm.

She seems to be doing well until the third day post-operatively when she begins to get drowsy and confused. Urea and electrolytes on her day of admission and her third post-operative day are reviewed and a one litre a day fluid restriction is instituted. However, her clinical condition fails to improve and further tests are carried out.

The summary of results is as follows:

	Day 1	Day 3	Day 4	Normal Range
Plasma sodium	136 mmol/L	113 mmol/L	114 mmol/L	(137-144)
Plasma potassium	4.9 mmol/L	3.5 mmol/L	3.3 mmol/L	(3.5-4.9)
Plasma urea	6.5 mmol/L	2.8 mmol/L	2.8 mmol/L	(2.5-7.5)
24 hour urinary sodium	70 mmol	-	-	-
Urine osmolality	-	-	710 mmol/kg H_2O	(350-1000)

What is the most likely cause of this patient's confusion?

(Please select 1 option)

○ Addison's disease

○ Syndrome of inappropriate ADH secretion

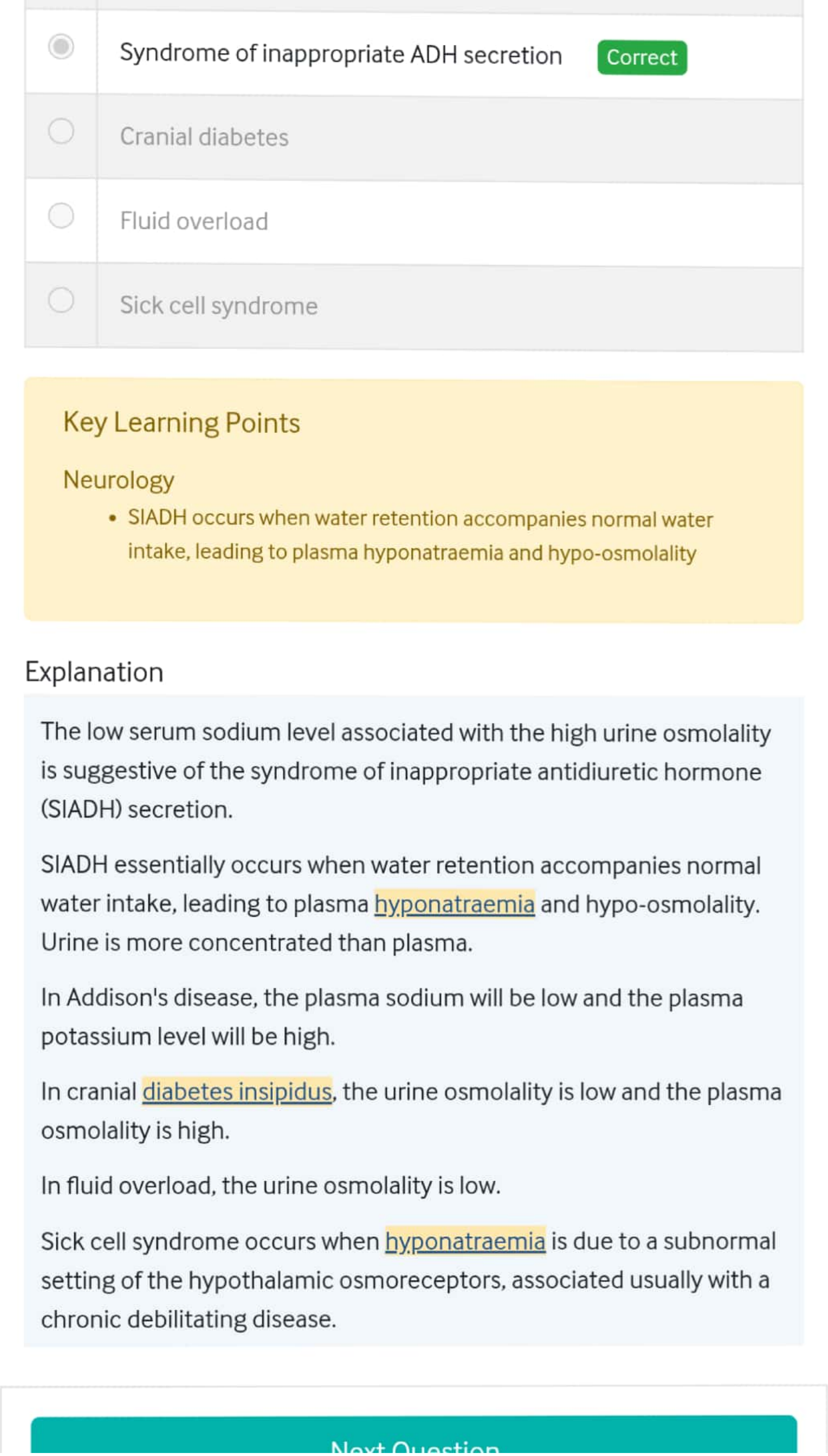
○ Cranial diabetes

○ Fluid overload

○ Sick cell syndrome

Submit Answer

CamScanner الممسوحة ضوئيا بـ



● Syndrome of inappropriate ADH secretion **Correct**

○ Cranial diabetes

○ Fluid overload

○ Sick cell syndrome

Key Learning Points

Neurology

- SIADH occurs when water retention accompanies normal water intake, leading to plasma hyponatraemia and hypo-osmolality

Explanation

The low serum sodium level associated with the high urine osmolality is suggestive of the syndrome of inappropriate antidiuretic hormone (SIADH) secretion.

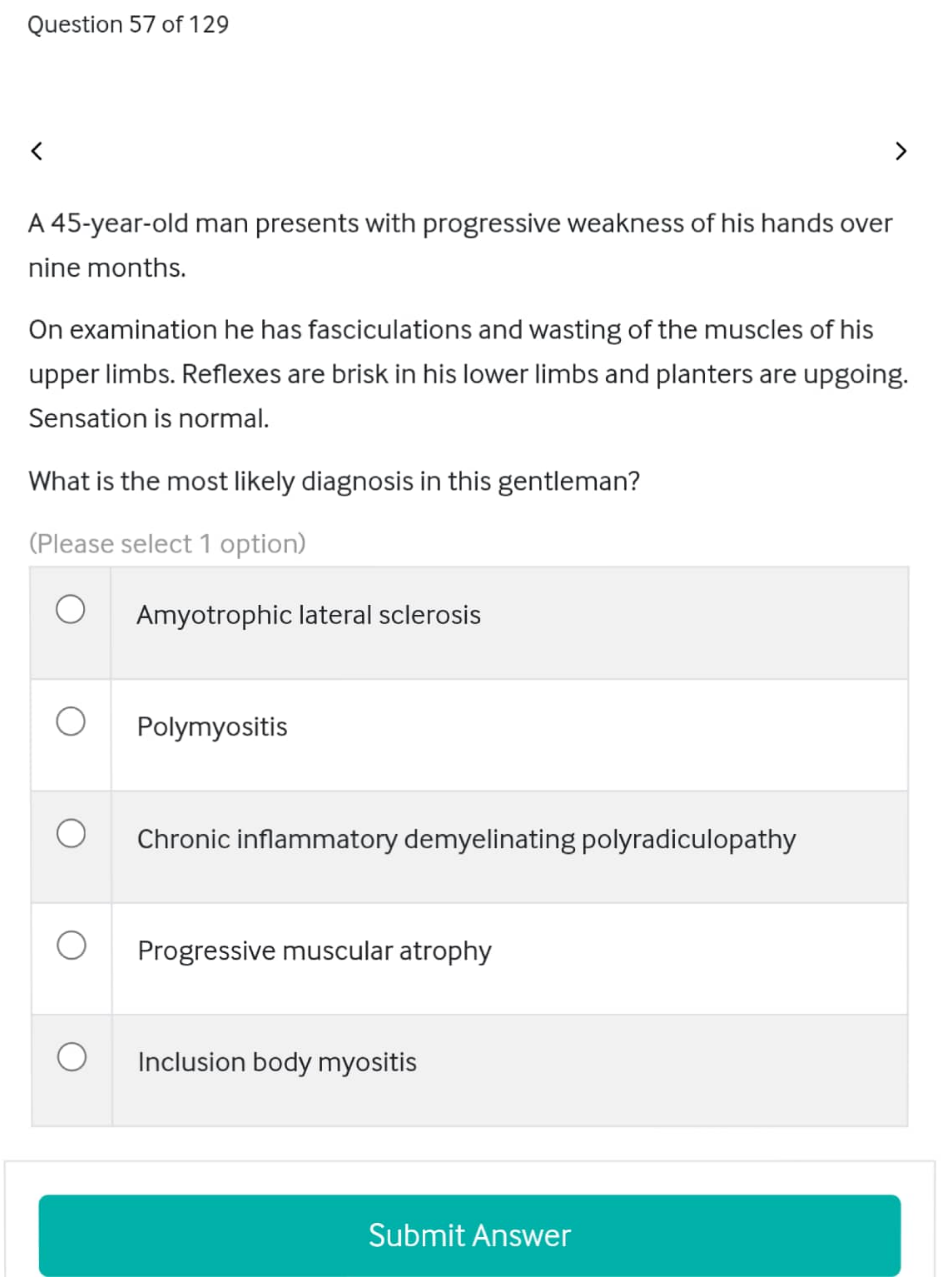
SIADH essentially occurs when water retention accompanies normal water intake, leading to plasma hyponatraemia and hypo-osmolality. Urine is more concentrated than plasma.

In Addison's disease, the plasma sodium will be low and the plasma potassium level will be high.

In cranial diabetes insipidus, the urine osmolality is low and the plasma osmolality is high.

In fluid overload, the urine osmolality is low.

Sick cell syndrome occurs when hyponatraemia is due to a subnormal setting of the hypothalamic osmoreceptors, associated usually with a chronic debilitating disease.



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A 45-year-old man presents with progressive weakness of his hands over nine months.

On examination he has fasciculations and wasting of the muscles of his upper limbs. Reflexes are brisk in his lower limbs and planters are upgoing. Sensation is normal.

What is the most likely diagnosis in this gentleman?

(Please select 1 option)

- ○ Amyotrophic lateral sclerosis
- ○ Polymyositis
- ○ Chronic inflammatory demyelinating polyradiculopathy
- ○ Progressive muscular atrophy
- ○ Inclusion body myositis

● Amyotrophic lateral sclerosis **Correct**

○ Polymyositis

○ Chronic inflammatory demyelinating polyradiculopathy

○ Progressive muscular atrophy

○ Inclusion body myositis

Key Learning Points

Neurology

- ALS is a mixture of lower motor neurone signs in the upper arms and upper motor neurone signs in the legs.

Explanation

There is a mixture of lower motor neurone signs in the upper arms and upper motor neurone signs in the legs. This patient has motor neurone disease of the amyotrophic lateral sclerosis type.

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an acquired immune mediated inflammatory disorder of the peripheral nervous system and is closely related to Guillain-Barré syndrome.

Patients usually present with a history of weakness, numbness and difficulty walking.

Inclusion body myositis (IBM) is an inflammatory disease resulting in slowly progressive weakness and wasting of both distal and proximal muscles.

Polymyositis is an inflammatory myopathy. Muscles may be painful and CK is markedly elevated.

Progressive muscular atrophy is a rare form of motor neurone disease which affects only the lower motor neurones resulting in fasciculations and progressive wasting of upper and lower limbs.

You are asked to see a 40-year-old woman who has a six week history of episodes of 'the room spinning around' lasting several seconds. It is typically most severe when she is lying on her right side with the ear down. During these episodes she also has nausea. She denies any problems with hearing or tinnitus.

On examination, she has a rotatory nystagmus with an onset several seconds after the sudden assumption of her head in the right lateral decubitus position using the Hallpike manoeuvre and which resolves after a minute or so.

Repeated positioning of the head in the precipitating position results in the attenuation of the nystagmus. No other abnormalities are detected.

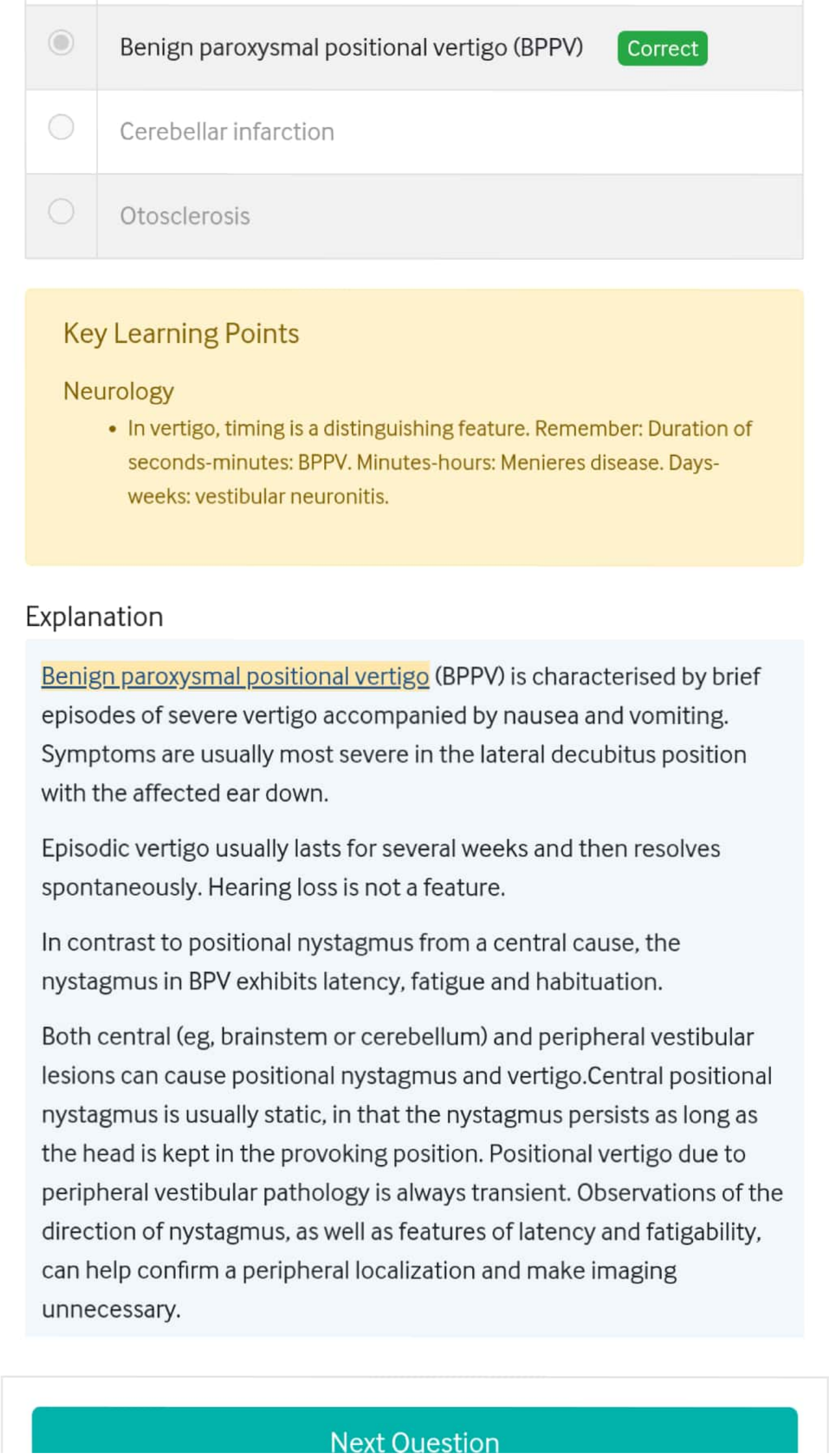
Audiometry investigation showed normal hearing.

What is this patient's likely diagnosis?

(Please select 1 option)

- ○ Cerebellopontine angle tumour
- ○ Meniere's disease
- ○ Benign paroxysmal positional vertigo (BPPV)
- ○ Cerebellar infarction
- ○ Otosclerosis

Submit Answer



● Benign paroxysmal positional vertigo (BPPV) Correct

○ Cerebellar infarction

○ Otosclerosis

Key Learning Points

Neurology

- In vertigo, timing is a distinguishing feature. Remember: Duration of seconds-minutes: BPPV. Minutes-hours: Menieres disease. Days-weeks: vestibular neuronitis.

Explanation

Benign paroxysmal positional vertigo (BPPV) is characterised by brief episodes of severe vertigo accompanied by nausea and vomiting. Symptoms are usually most severe in the lateral decubitus position with the affected ear down.

Episodic vertigo usually lasts for several weeks and then resolves spontaneously. Hearing loss is not a feature.

In contrast to positional nystagmus from a central cause, the nystagmus in BPV exhibits latency, fatigue and habituation.

Both central (eg, brainstem or cerebellum) and peripheral vestibular lesions can cause positional nystagmus and vertigo.Central positional nystagmus is usually static, in that the nystagmus persists as long as the head is kept in the provoking position. Positional vertigo due to peripheral vestibular pathology is always transient. Observations of the direction of nystagmus, as well as features of latency and fatigability, can help confirm a peripheral localization and make imaging unnecessary.

CamScanner المسوحة ضوئيا بـ

A 22-year-old lady presented to the Emergency department having had a tonic-clonic seizure whilst in the shopping centre. She had not complained of feeling unwell prior to the event.

She had a past medical history of learning difficulties and had been referred to a dermatologist in the last year with lesions over her face. Otherwise she took no regular medication, did not smoke or drink alcohol.

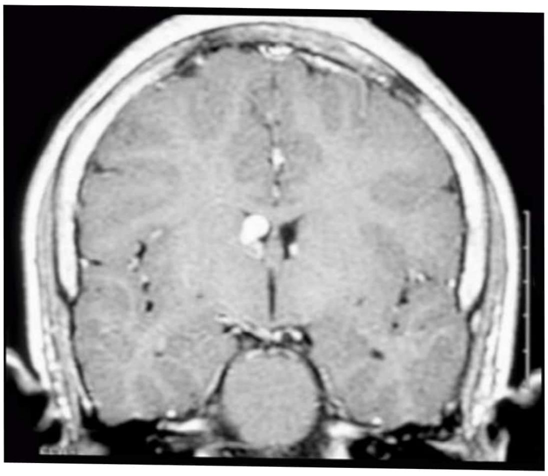
On examination she was post-ictal. Her vital signs were stable. She had pink nodules with a smooth glistening surface affecting the cheeks and nasolabial folds. In addition, there were elevated skin-coloured lesions over the trunk.

Fundoscopy revealed bilateral retinal phakomas, but the rest of the cranial nerves were intact. Peripheral nervous system examination demonstrated bilaterally brisk reflexes with bilateral extensor plantar responses.

Investigations revealed:

Haemoglobin	146 g/L	(130-180)
Mean cell volume	92 fL	(80-96)
White cell count	6.1 $\times 10^9$/L	(4-11)
Platelets	239 $\times 10^9$/L	(150-400)
Serum sodium	139 mmol/L	(137-144)
Serum potassium	4.6 mmol/L	(3.5-4.9)
Serum urea	4.8 mmol/L	(2.5-7.5)
Serum creatinine	95 µmol/L	(60-110)
Serum calcium	2.4 mmol/L	(2.2-2.6)
Serum C reactive protein	<5 U/L	(<5)
Serum albumin	39 g/L	(37-49)
Fasting plasma glucose	5.6 mmol/L	(3-6)

A CT scan of the brain was performed. A pre-contrast image from this study is shown below:



What is the most likely diagnosis?

(Please select 1 option)

○ Multiple endocrine neoplasia type Ib

○ Neurofibromatosis

○ Tuberous sclerosis

○ Gardner's syndrome

○ von Hippel-Lindau syndrome

Submit Answer

CamScanner المسوحة ضوئيا بـ

○ Neurofibromatosis

● Tuberous sclerosis **Correct**

○ Gardner's syndrome

○ von Hippel-Lindau syndrome

Key Learning Points

Dermatology, Genetics, Neurology, Photographic

- Tuberous sclerosis presents with flesh coloured papules (angiofibromas) on the face and fibromas in the central nervous system

Explanation

This patient has tuberous sclerosis.

Fibromas may also develop within the central nervous system, where they calcify typically in the periventricular area.

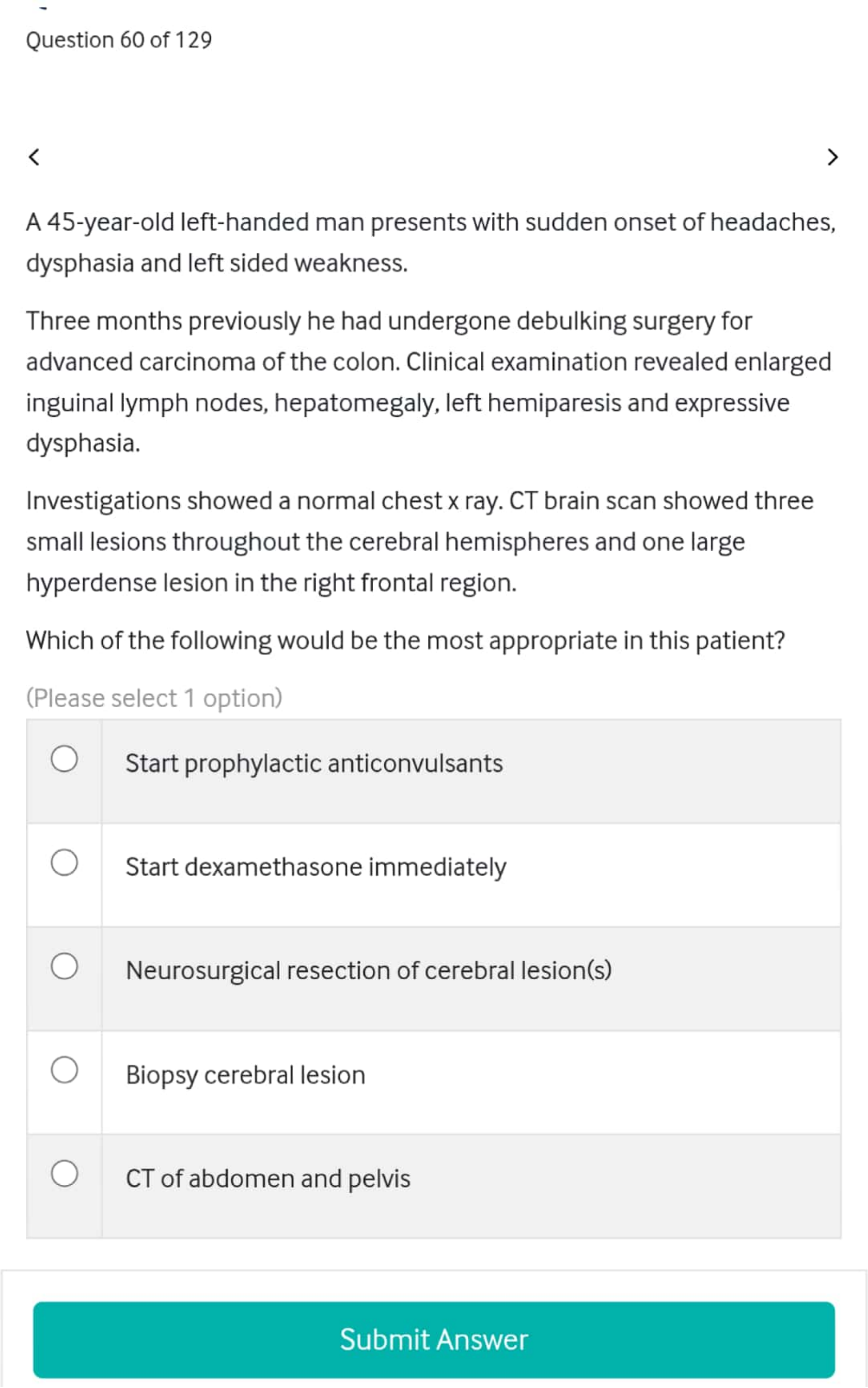
Other manifestations of tuberose sclerosis include

- Ash-leaf macules, which are discrete areas of hypopigmentation, often present at birth
- Shagreen patch in the form of a rough patch of skin with a texture similar to shark skin and
- Adenoma sebaceum, which is angiofibromas of the face.

Reference:

Tuberous Sclerosis Association: Factsheets

Next Question



A 45-year-old left-handed man presents with sudden onset of headaches, dysphasia and left sided weakness.

Three months previously he had undergone debulking surgery for advanced carcinoma of the colon. Clinical examination revealed enlarged inguinal lymph nodes, hepatomegaly, left hemiparesis and expressive dysphasia.

Investigations showed a normal chest x ray. CT brain scan showed three small lesions throughout the cerebral hemispheres and one large hyperdense lesion in the right frontal region.

Which of the following would be the most appropriate in this patient?

(Please select 1 option)

- ○ Start prophylactic anticonvulsants
- ○ Start dexamethasone immediately
- ○ Neurosurgical resection of cerebral lesion(s)
- ○ Biopsy cerebral lesion
- ○ CT of abdomen and pelvis

● Start dexamethasone immediately **Correct**

○ Neurosurgical resection of cerebral lesion(s)

○ Biopsy cerebral lesion

○ CT of abdomen and pelvis

Key Learning Points

Neurology

- Dexamethasone helps to reduce swelling around malignant lesions and can help to relieve compression symptoms.

Explanation

This patient has definitive multiple cerebral secondaries (the hyperdense lesion on CT suggests intratumoural bleed) and aggressive primary disease.

The extent of the disease excludes the need for further investigations.

The treatment of choice in this case is dexamethasone which must be started immediately, alongside proton pump inhibitor or an H2 antagonist to protect the stomach and bone protection.

Followed by this a staging CT is required and then discussion in an appropriate multi-disciplinary team setting.

Anticonvulsant prophylaxis is unnecessary if seizures are not a problem and are not effective in preventing first seizures in patients with newly diagnosed brain tumours.

Surgical resection is usually only recommended if a single cerebral lesion is found.

CamScanner الممسوحة ضوئيا بـ

A 62-year-old man presented with difficult walking.

He had a past history of diabetes mellitus and cervical spondylosis, which had required surgical decompression eight years previously. He drank 40 units of alcohol weekly.

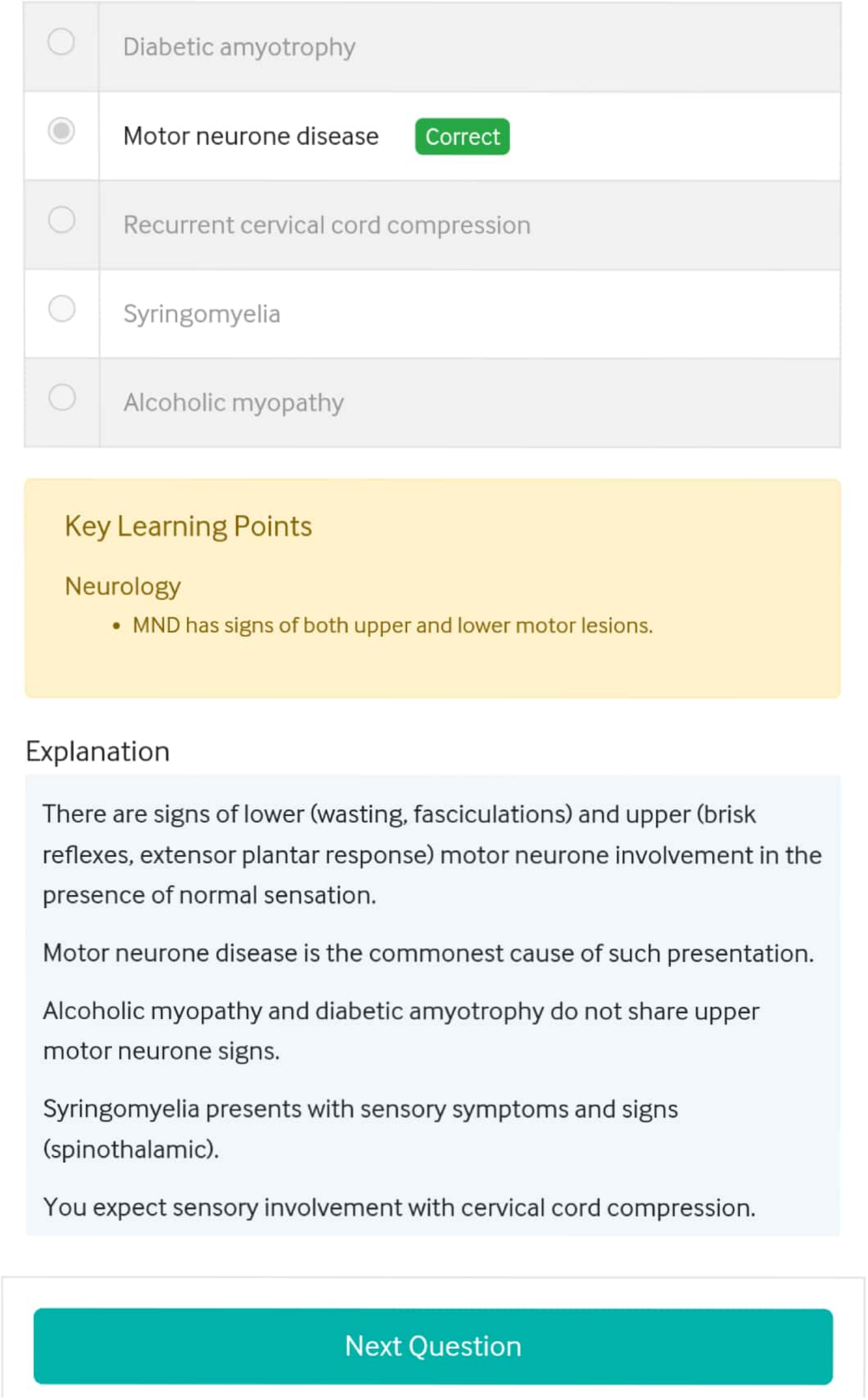
On examination there was fasciculation, wasting and weakness in the left deltoid and biceps, with weakness in the shoulder girdle muscles bilaterally. There was fasciculation in the glutei and quadriceps bilaterally, weakness of hip flexion and foot dorsiflexion, brisk reflexes in upper and lower limbs, and extensor plantar responses. There was no sensory impairment.

What is the diagnosis?

(Please select 1 option)

- ○ Diabetic amyotrophy
- ○ Motor neurone disease
- ○ Recurrent cervical cord compression
- ○ Syringomyelia
- ○ Alcoholic myopathy

Submit Answer



○ Diabetic amyotrophy

● Motor neurone disease **Correct**

○ Recurrent cervical cord compression

○ Syringomyelia

○ Alcoholic myopathy

Key Learning Points

Neurology

- MND has signs of both upper and lower motor lesions.

Explanation

There are signs of lower (wasting, fasciculations) and upper (brisk reflexes, extensor plantar response) motor neurone involvement in the presence of normal sensation.

Motor neurone disease is the commonest cause of such presentation.

Alcoholic myopathy and diabetic amyotrophy do not share upper motor neurone signs.

Syringomyelia presents with sensory symptoms and signs (spinothalamic).

You expect sensory involvement with cervical cord compression.

Question 62 of 129

‹ ›

A 41-year-old woman presented to hospital with a two day history of severe right side headache and loss of vision.

She had a past history of hypertension, for which she was taking bendroflumethiazide. There was a family history of 'thyroid problems'. There was a past history of intermittent headaches, though these were not regular. Her current headache was the worst she had ever experienced.

On examination she was agitated and distressed. Her temperature was 37.5°C, pulse 110 beats per minute and blood pressure 145/95 mmHg. There was chemosis and proptosis of the right eye. Upward and downward gaze was paralysed; lateral gaze was preserved. The right pupil was dilated and unresponsive to light. The left eye was normal. Fundoscopy showed bilateral papilloedema.

What is the most likely diagnosis?

(Please select 1 option)

○ Cavernous sinus thrombosis

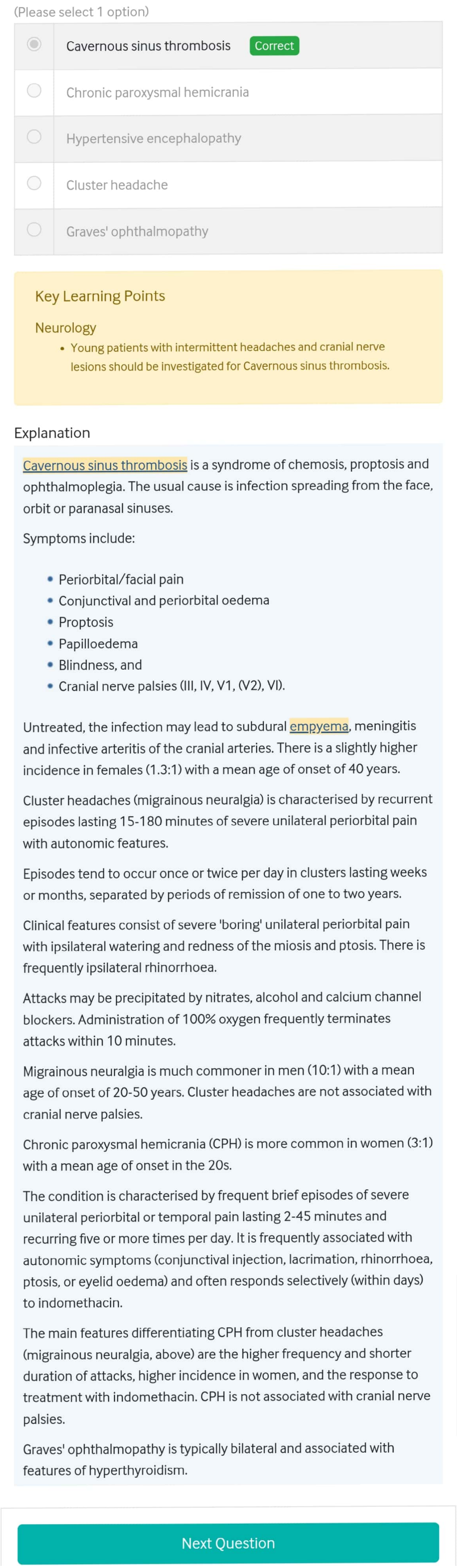
○ Chronic paroxysmal hemicrania

○ Hypertensive encephalopathy

○ Cluster headache

○ Graves' ophthalmopathy

Submit Answer



(Please select 1 option)

- ● Cavernous sinus thrombosis **Correct**
- ○ Chronic paroxysmal hemicrania
- ○ Hypertensive encephalopathy
- ○ Cluster headache
- ○ Graves' ophthalmopathy

Key Learning Points

Neurology

- Young patients with intermittent headaches and cranial nerve lesions should be investigated for Cavernous sinus thrombosis.

Explanation

Cavernous sinus thrombosis is a syndrome of chemosis, proptosis and ophthalmoplegia. The usual cause is infection spreading from the face, orbit or paranasal sinuses.

Symptoms include:

- Periorbital/facial pain
- Conjunctival and periorbital oedema
- Proptosis
- Papilloedema
- Blindness, and
- Cranial nerve palsies (III, IV, V1, (V2), VI).

Untreated, the infection may lead to subdural empyema, meningitis and infective arteritis of the cranial arteries. There is a slightly higher incidence in females (1.3:1) with a mean age of onset of 40 years.

Cluster headaches (migrainous neuralgia) is characterised by recurrent episodes lasting 15-180 minutes of severe unilateral periorbital pain with autonomic features.

Episodes tend to occur once or twice per day in clusters lasting weeks or months, separated by periods of remission of one to two years.

Clinical features consist of severe 'boring' unilateral periorbital pain with ipsilateral watering and redness of the miosis and ptosis. There is frequently ipsilateral rhinorrhoea.

Attacks may be precipitated by nitrates, alcohol and calcium channel blockers. Administration of 100% oxygen frequently terminates attacks within 10 minutes.

Migrainous neuralgia is much commoner in men (10:1) with a mean age of onset of 20-50 years. Cluster headaches are not associated with cranial nerve palsies.

Chronic paroxysmal hemicrania (CPH) is more common in women (3:1) with a mean age of onset in the 20s.

The condition is characterised by frequent brief episodes of severe unilateral periorbital or temporal pain lasting 2-45 minutes and recurring five or more times per day. It is frequently associated with autonomic symptoms (conjunctival injection, lacrimation, rhinorrhoea, ptosis, or eyelid oedema) and often responds selectively (within days) to indomethacin.

The main features differentiating CPH from cluster headaches (migrainous neuralgia, above) are the higher frequency and shorter duration of attacks, higher incidence in women, and the response to treatment with indomethacin. CPH is not associated with cranial nerve palsies.

Graves' ophthalmopathy is typically bilateral and associated with features of hyperthyroidism.

A 39-year-old HIV seropositive man was referred to the neurology clinic with altered personality and progressive speech problems.

His partner, who had accompanied him to clinic, said that he had noticed a marked difference in his demeanour and that he had become aggressive towards him in the last few weeks. The patient had also noticed increasing stiffness in his legs and had been dragging his left leg whilst walking.

The patient was diagnosed with HIV approximately 10 years ago. He had been offered the opportunity to start antiretroviral therapy several times, but had declined due to his concerns about the possible side-effects of drugs. Otherwise he had no other past medical history, was a smoker of 10 cigarettes per day and drank 20 units of alcohol per week.

On examination he appeared thin and drawn. He weighed 50 kg in his clothes. His temperature was 36.1°C, blood pressure was 135/75 mmHg and pulse was 78/minute and regular. He had several cold sores around his mouth. He was alert and orientated. He scored 27/30 on mini-mental state examination with deficiencies in naming objects. Cranial nerve examination was normal.

On examination of the peripheral nervous system, there was a mild left-sided hemiparesis and the right plantar reflex was extensor. He walked with a spastic gait and appeared to be leaning to the left.

The results of further investigations are detailed below:

MRI brain scan: Multiple, non-enhancing white-matter lesions within the frontal and temporal lobes with no oedema or mass-effect.

Blood tests showed:

Serum oligoclonal bands	Present
CD4 count	96 cells/mm^3
HIV quantitative PCR (viral load)	100,000 copies/ml

Lumbar puncture showed:

Opening pressure	16 cm H_2O	(5-18)
CSF protein	0.42 g/L	(0.15 - 0.45)
CSF white cell count	4 per ml	(5)
CSF red cell count	3 per ml	(5)
CSF oligoclonal bands	Present	-

What is the most likely diagnosis?

(Please select 1 option)

○	Progressive multifocal leukoencephalopathy
○	HIV encephalopathy
○	CNS lymphoma
○	*Toxoplasma* encephalitis
○	Primary progressive multiple sclerosis (MS)

Submit Answer

(Please select 1 option)

● Progressive multifocal leukoencephalopathy Correct

○ HIV encephalopathy

○ CNS lymphoma

○ *Toxoplasma* encephalitis

○ Primary progressive multiple sclerosis (MS)

Key Learning Points

Neurology

- Progressive multifocal leukoencephalopathy (PML) is the result of infection of oligodendrocytes with JC virus and common in patients with HIV and low CD4 counts.

Explanation

This patient presents with a left hemiparesis, expressive dysphasia and behavioural changes in clear consciousness.

The most likely diagnosis is progressive multifocal leukoencephalopathy.

Progressive multifocal leukoencephalopathy (PML) is the result of infection of oligodendrocytes with JC virus, which is a papovavirus found latent in most healthy adults. With CD4 counts of less than 100 this virus becomes active leading to progressive neurological deterioration. Hemiparesis, visual field defects, ataxia and mental change are characteristic.

The diagnosis can be established either

- By demonstrating the presence of JC virus genetic material in the cerebrospinal fluid (by polymerase chain reaction), or
- Through a brain biopsy (showing asymmetric foci of demyelination and intranuclear inclusions containing the JC virus).

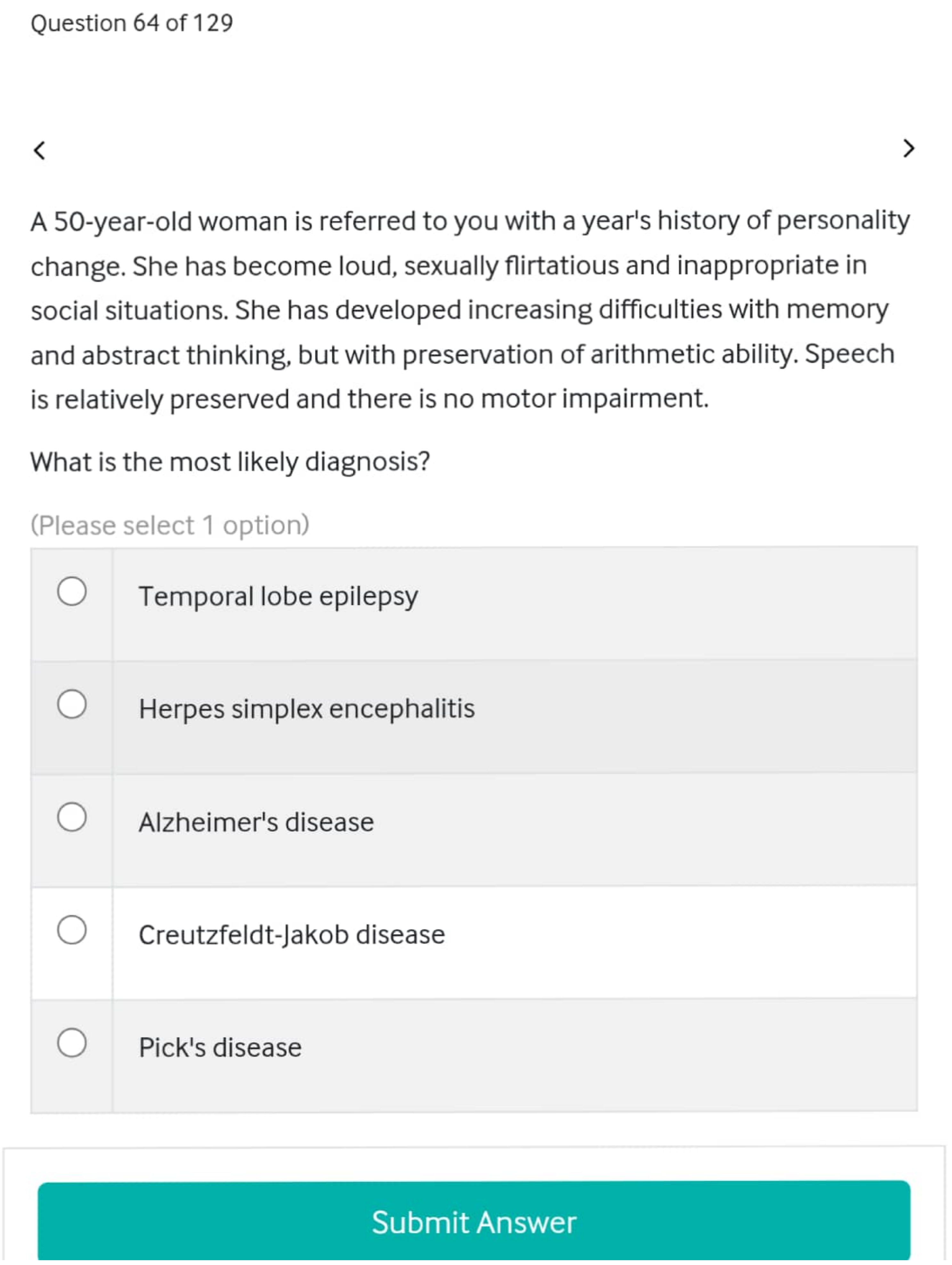
There is no effective treatment, but progression can be slowed by initiation of antiretroviral therapy.

Primary central nervous system lymphoma usually presents with a solitary space-occupying lesion with mass effect.

Toxoplasma encephalitis typically presents as multiple ring-enhancing lesions with mass effect; the patient is often febrile and the presentation is usually more acute.

HIV encephalopathy is frequently associated with behavioural changes, but radiological changes are usually limited to cerebral atrophy.

In primary progressive MS there is intrathecal oligoclonal band synthesis only, and the white matter lesions are periventricular in location.



Question 64 of 129

A 50-year-old woman is referred to you with a year's history of personality change. She has become loud, sexually flirtatious and inappropriate in social situations. She has developed increasing difficulties with memory and abstract thinking, but with preservation of arithmetic ability. Speech is relatively preserved and there is no motor impairment.

What is the most likely diagnosis?

(Please select 1 option)

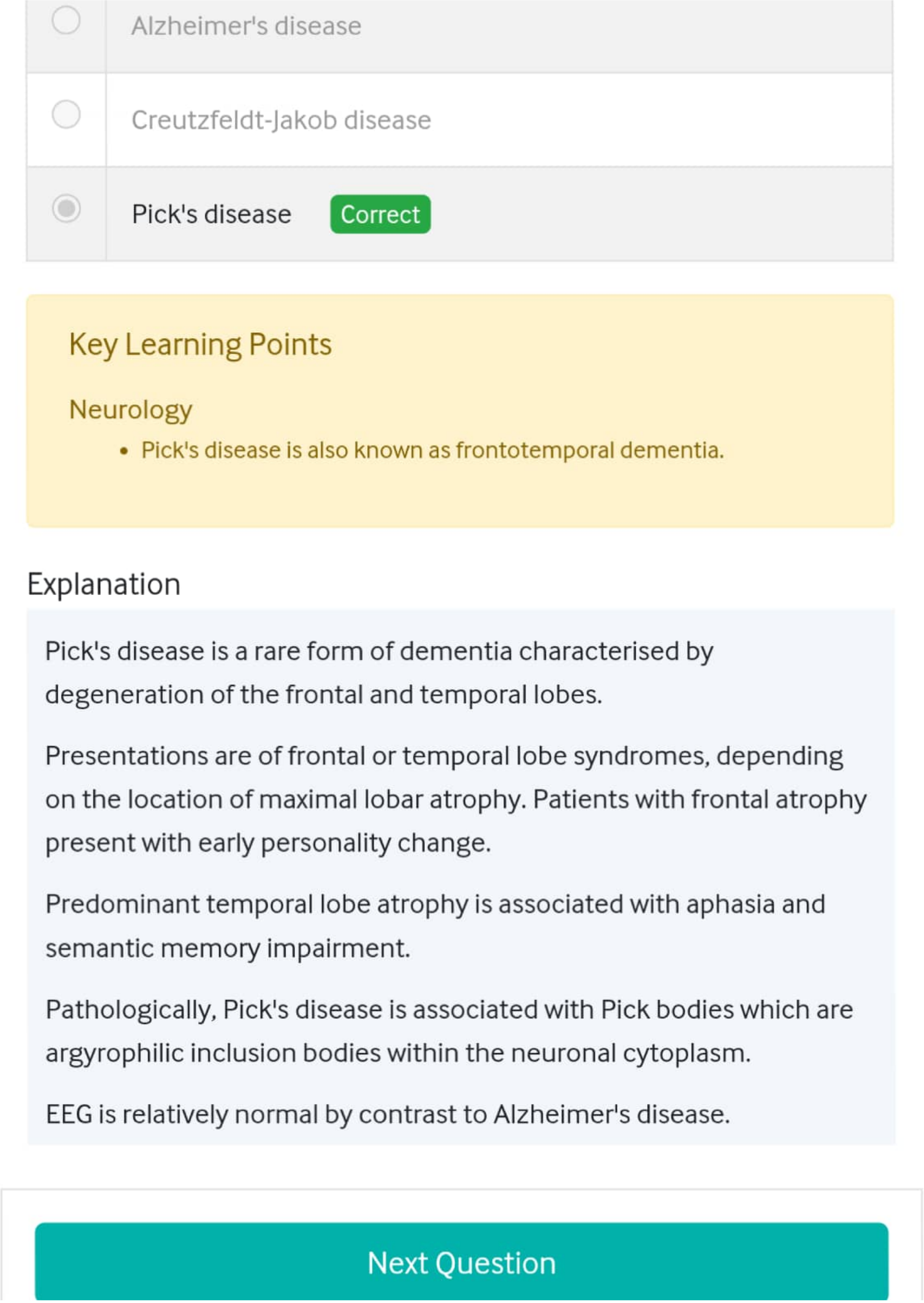
○ Temporal lobe epilepsy

○ Herpes simplex encephalitis

○ Alzheimer's disease

○ Creutzfeldt-Jakob disease

○ Pick's disease



○ Alzheimer's disease

○ Creutzfeldt-Jakob disease

● Pick's disease Correct

Key Learning Points

Neurology

- Pick's disease is also known as frontotemporal dementia.

Explanation

Pick's disease is a rare form of dementia characterised by degeneration of the frontal and temporal lobes.

Presentations are of frontal or temporal lobe syndromes, depending on the location of maximal lobar atrophy. Patients with frontal atrophy present with early personality change.

Predominant temporal lobe atrophy is associated with aphasia and semantic memory impairment.

Pathologically, Pick's disease is associated with Pick bodies which are argyrophilic inclusion bodies within the neuronal cytoplasm.

EEG is relatively normal by contrast to Alzheimer's disease.

A 70-year-old diabetic man presented with a two-hour history of mild right-sided hand weakness. He gave a history of several transient ischaemic attacks over the past year in spite of taking aspirin daily. He was also taking metformin and gliclazide and a diabetic diet for glycaemic control.

On examination, his BMI was 32.2 kg/m^2, his blood pressure was 152/96 mmHg and his pulse was 88 beats per minute and regular. Cardiovascular examination revealed no abnormalities on auscultation over the praecordium, but a left sided carotid bruit was heard. Neurological examination on admission had revealed a right hemiparesis and an extensor right plantar response. One hour later there was weakness in the right arm and a flexor right plantar response. There were no other abnormalities of note.

Investigations revealed:

Full blood count	Normal	
U+Es	Normal	
Liver function	Normal	
Brain CT scan	Normal	
Random blood glucose	26.5 mmol/L	(<11)
Prothrombin time	14 secs	(11.5-15.5)

Which of the following would be contraindications to thrombolytic treatment?

(Please select 1 option)

- ○ Aspirin therapy
- ○ Resolving neurological signs
- ○ The presence of a carotid bruit
- ○ His blood pressure
- ○ The prothrombin time

Submit Answer

(Please select 1 option)

○ Aspirin therapy

● Resolving neurological signs **Correct**

○ The presence of a carotid bruit

○ His blood pressure

○ The prothrombin time

Key Learning Points

Neurology

- Resolving neurological signs are a contraindication to thrombolysis.

Explanation

Questions about thromolytic therapy are popular, so knowledge of the indications and contraindications to thrombolytic theory are important.

There are risks associated with thrombolytic therapy, the greatest being IC bleed.

Further Reading:

Patient.info. Thrombolytic Treatment of Acute Ischaemic Stroke.

Next Question

Question 66 of 129

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A 60-year-old man presents with a two month history of progressive confusion, gait disturbance, and urinary incontinence.

Examination reveals mild global cognitive dysfunction and gait ataxia.

CT brain shows enlarged ventricles with no evidence of obstruction to CSF outflow. Lumbar puncture reveals normal CSF pressure and constituents.

Which one of the following managements steps is likely to be most helpful?

(Please select 1 option)

○	Serum B12 and folate levels
○	CSF drainage via repeated lumbar puncture
○	MRI brainstem
○	EEG
○	Intracranial pressure monitoring

Submit Answer

● CSF drainage via repeated lumbar puncture **Correct**

○ MRI brainstem

○ EEG

○ Intracranial pressure monitoring

Key Learning Points

Neurology, Psychiatry

- Normal pressure hydrocephalus is treated with repeated therapeutic lumbar puncture.

Explanation

Normal pressure hydrocephalus presents with the triad of:

- Dementia
- Gait disturbance and
- Incontinence.

It is associated with episodes of increased pressure related to impaired cerebrospinal fluid (CSF) absorption and therefore may benefit from CSF shunting.

Although the ventricular enlargement described in this case may derive from global atrophy and need not represent hydrocephalus the other features of the case strongly point to the diagnosis.

In equivocal cases therapeutic CSF drainage via lumbar puncture is performed to identify the patients likely to benefit from permanent drainage.

Intracranial pressure monitoring to demonstrate the episodes of increased pressure is a more invasive alternative.

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A 70-year-old man presented with 12 months history of fluctuating memory and agitation. He also complains of visual hallucinations. His family were concerned and had taken him to see his GP who had prescribed him haloperidol which he had been taking for the last two months but this was withdrawn recently as the patient became more unwell.

On examination, he had bilateral limb rigidity and bradykinesia, and a mild right-sided rest tremor. The rest of the neurological examination was normal.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Alzeimer’s disease
- ○ Progressive supranuclear palsy
- ○ Multiple system atrophy
- ○ Diffuse Lewy body disease
- ○ Idiopathic Parkinson’s disease

Submit Answer

○ Progressive supranuclear palsy

○ Multiple system atrophy

● Diffuse Lewy body disease **Correct**

○ Idiopathic Parkinson’s disease

Key Learning Points

Neurology, Psychiatry

- Diffuse Lewy body disease is characterised by fluctuating cognitive impairment, visual hallucinations and parkinsonism.

Explanation

Diffuse Lewy body disease is characterised by fluctuating cognitive impairment, visual hallucinations and parkinsonism.

The patients are very sensitive to neuroleptics which therefore should be avoided.

The treatment of choice is rivastigmine, which improves both the visual hallucinations, and cognitive impairment.[1]

A 59-year-old right handed woman was referred to neurology clinic having developed tingling affecting her right hand. The tingling appeared worse at night and she had disrupted sleep as a result often needing to shake her hand to get rid of the tingling. She had also noticed recent problems with grip particularly when manipulating fine objects or unscrewing jam jar lids and was also beginning to experience an aching sensation radiating up the forearm. Sometimes she commented that her fingers felt swollen.

She had a past medical history of hypothyroidism and diabetes, and took regular thyroxine and metformin. She worked in a supermarket on the checkout tills and had noticed the problem since commencing work. She was a smoker of 10 cigarettes per day and drank 10 units of alcohol per week.

On examination there was some mild wasting of the right thenar eminence with some fasciculations. Tone was normal in the fingers. On examination of power there was reduced power of thumb opposition, abduction and flexion, with preserved interossei. Reflexes were all present. There was subtle sensory loss over the thumb, index, middle and lateral half of the ring finger extending to the wrist. Flexion at the wrist appeared to bring on tingling and numbness within this area.

Clinically she appeared to have a right carpal tunnel syndrome.

Nerve conduction studies of the right hand showed:

Median motor latency	5.5 milliseconds	(<4.5)
Median sensory latency	4.5 milliseconds	(<3.5)

Diminished compound muscle action potential and sensory nerve action potential amplitudes.

An EMG showed fibrillation potentials within the right abductor pollicis.

She was referred to orthopaedic surgery and offered an open carpal tunnel release and although it was explained that complications of surgery were rare with a 90% success rate, she declined this offer.

What might be a long term alternative treatment option for this patient?

(Please select 1 option)

○ Steroid injections

○ Laser acupuncture

○ Oral steroids

○ Bendroflumethiazide

○ Ultrasound therapy

Submit Answer

● Steroid injections **Correct**

○ Laser acupuncture

○ Oral steroids

○ Bendroflumethiazide

○ Ultrasound therapy

Key Learning Points

Neurology

- Ultrasound still needs to be compared to other treatments for CTS

Explanation

There are several non-surgical options for carpal tunnel syndrome; these non-surgical options were reviewed in the Journal of Neurology[1].

Diuretics, pyridoxine, non-steroidal anti-inflammatory drugs, yoga and laser-acupuncture seem to be ineffective in providing short term symptom relief (varying levels of evidence) and steroid injections seem to be effective (limited evidence).

There is conflicting evidence for the efficacy of ultrasound and oral steroids and given the fact that this patient is diabetic may be a less ideal treatment option. For providing long term relief from symptoms there is limited evidence that ultrasound is effective, and that splinting is less effective than surgery.

A combination of physiotherapy, splinting and steroid injections would be an effective non-surgical treatment regime for this patient.

There is a paucity of evidence surrounding use of ultrasound in carpal tunnel syndrome.

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A 70-year-old woman presents with paroxysmal dizziness where she has episodes where the room spins. These symptoms are exacerbated when she rolls over in bed to the right and when she reaches above her head. There are no other symptoms.

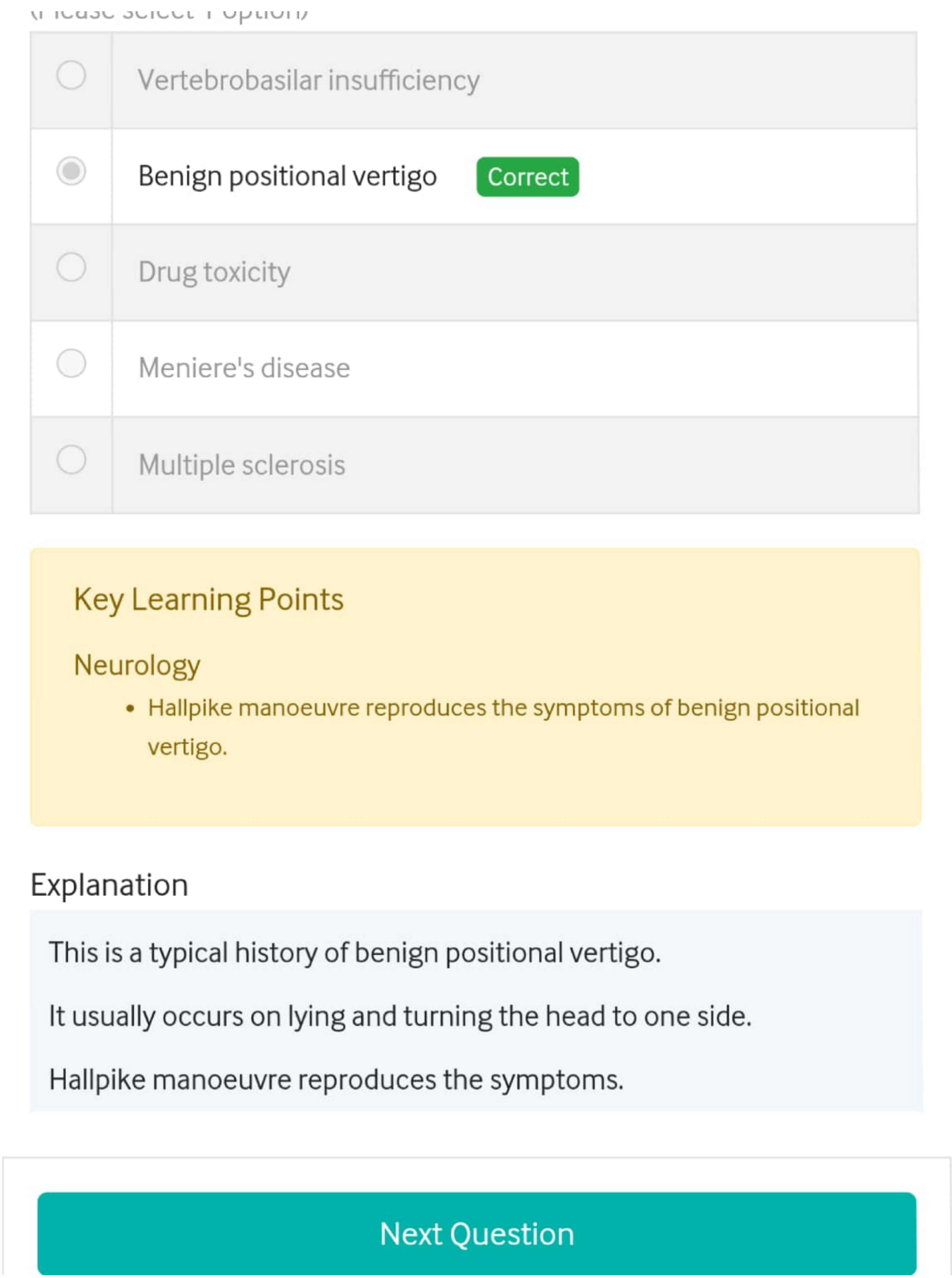
Neurological and general medical examination is otherwise normal. The vertigo can be reproduced by turning the head of the patient 45 degrees to the right and then taking the patient to the supine position. There is nystagmus (upbeating and torsional), which last only a few seconds.

What is the diagnosis?

(Please select 1 option)

○	Vertebrobasilar insufficiency
○	Benign positional vertigo
○	Drug toxicity
○	Meniere's disease
○	Multiple sclerosis

Submit Answer



(Please select 1 option)

○	Vertebrobasilar insufficiency
●	Benign positional vertigo **Correct**
○	Drug toxicity
○	Meniere's disease
○	Multiple sclerosis

Key Learning Points

Neurology

- Hallpike manoeuvre reproduces the symptoms of benign positional vertigo.

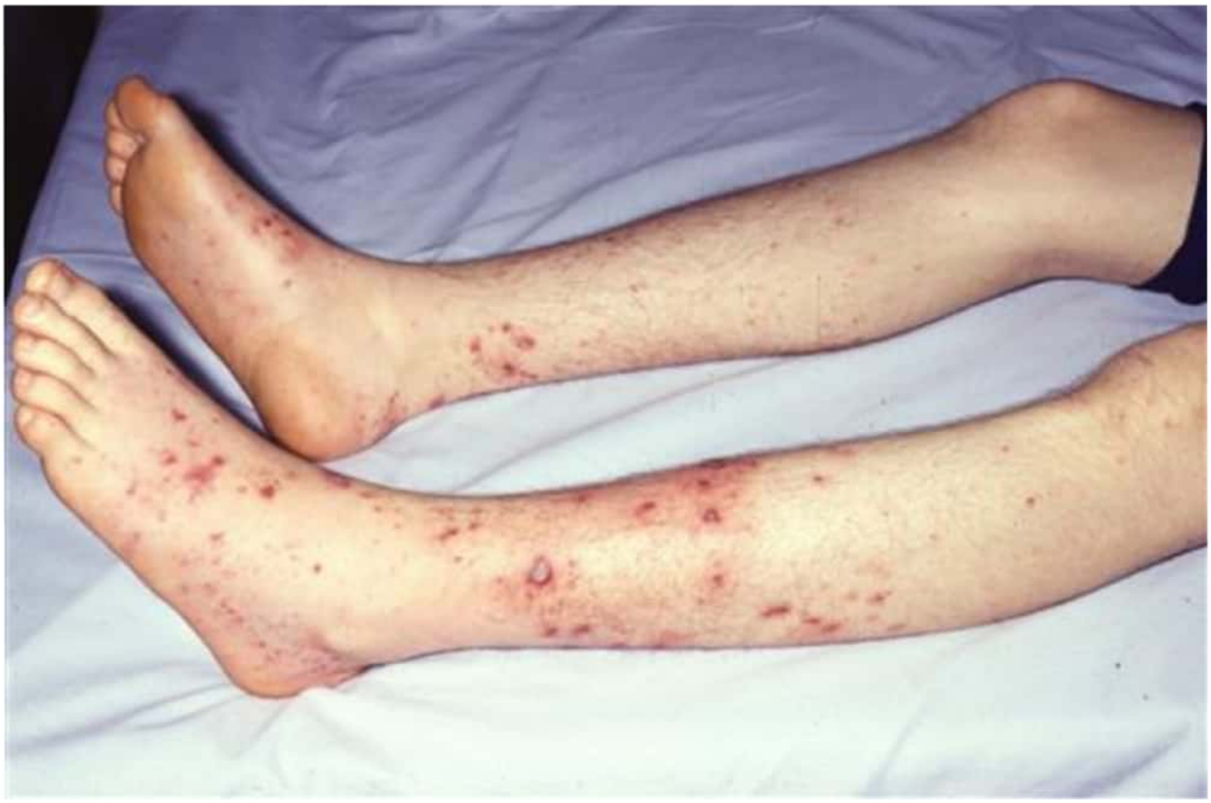
Explanation

This is a typical history of benign positional vertigo.

It usually occurs on lying and turning the head to one side.

Hallpike manoeuvre reproduces the symptoms.

This patient was brought in to hospital unresponsive with these initial findings.

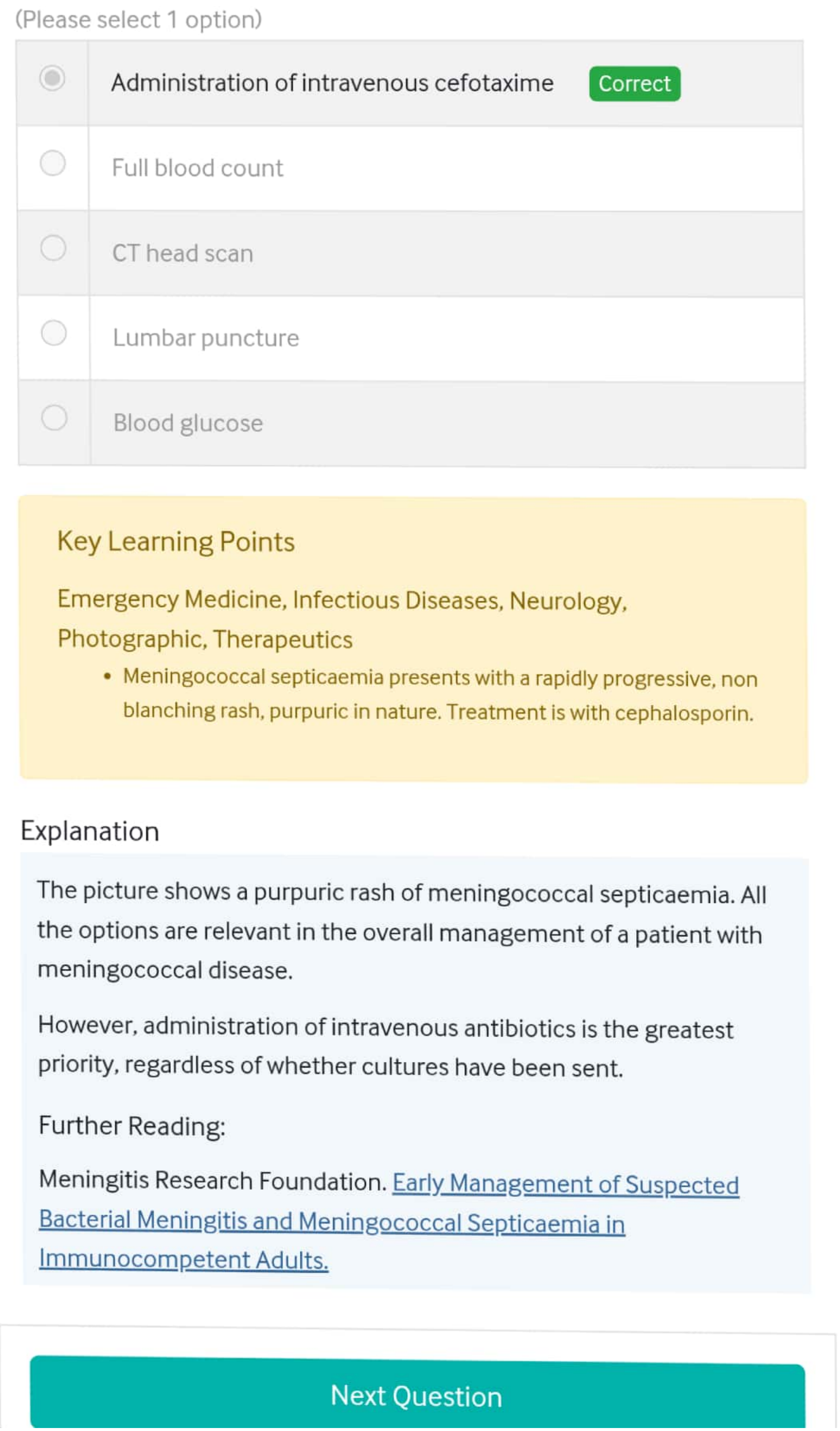


Which of the following is the most important initial step in management?

(Please select 1 option)

- ○ Administration of intravenous cefotaxime
- ○ Full blood count
- ○ CT head scan
- ○ Lumbar puncture
- ○ Blood glucose

Submit Answer



(Please select 1 option)

● Administration of intravenous cefotaxime **Correct**

○ Full blood count

○ CT head scan

○ Lumbar puncture

○ Blood glucose

Key Learning Points

Emergency Medicine, Infectious Diseases, Neurology, Photographic, Therapeutics

- Meningococcal septicaemia presents with a rapidly progressive, non blanching rash, purpuric in nature. Treatment is with cephalosporin.

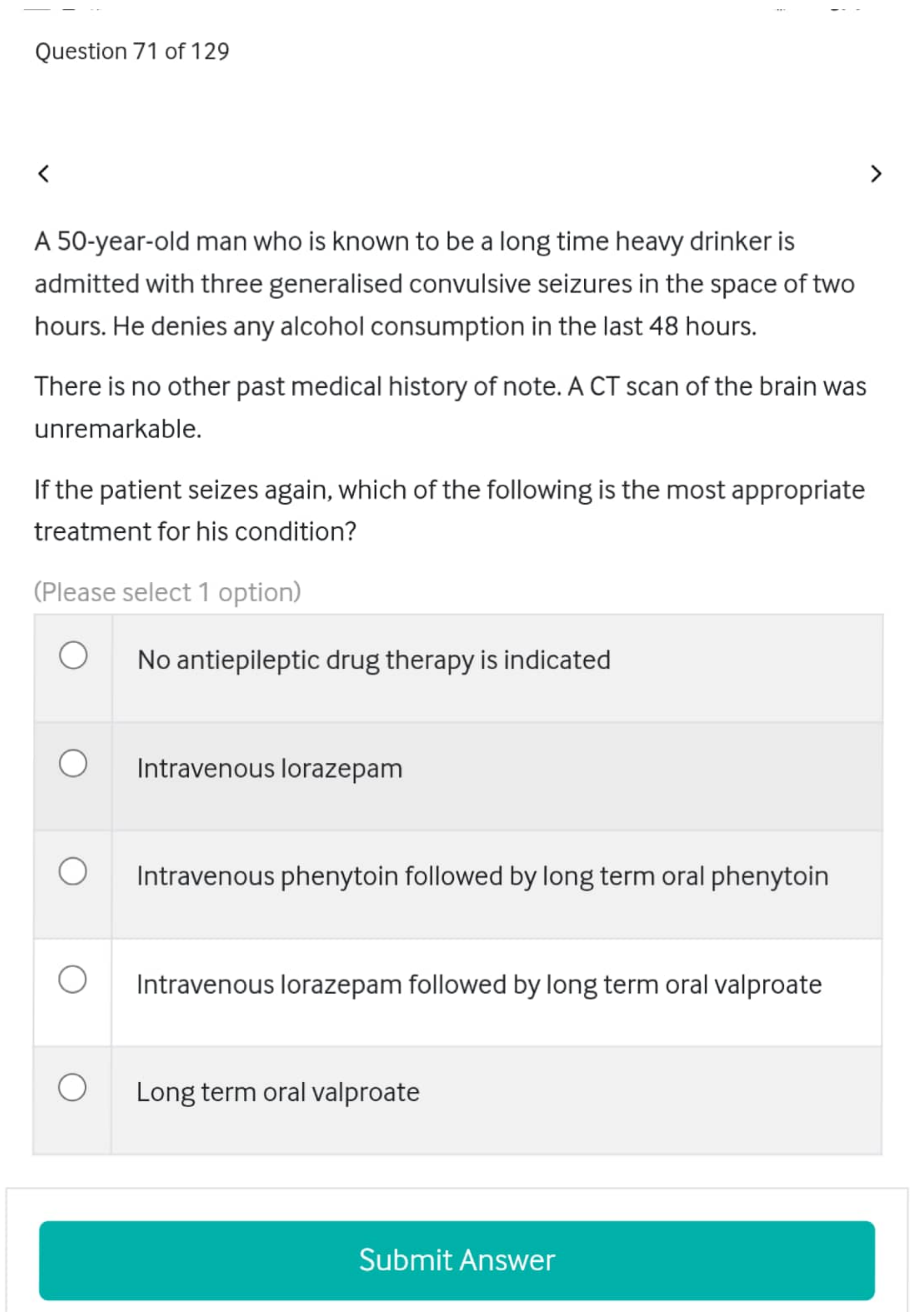
Explanation

The picture shows a purpuric rash of meningococcal septicaemia. All the options are relevant in the overall management of a patient with meningococcal disease.

However, administration of intravenous antibiotics is the greatest priority, regardless of whether cultures have been sent.

Further Reading:

Meningitis Research Foundation. Early Management of Suspected Bacterial Meningitis and Meningococcal Septicaemia in Immunocompetent Adults.



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A 50-year-old man who is known to be a long time heavy drinker is admitted with three generalised convulsive seizures in the space of two hours. He denies any alcohol consumption in the last 48 hours.

There is no other past medical history of note. A CT scan of the brain was unremarkable.

If the patient seizes again, which of the following is the most appropriate treatment for his condition?

(Please select 1 option)

- ○ No antiepileptic drug therapy is indicated
- ○ Intravenous lorazepam
- ○ Intravenous phenytoin followed by long term oral phenytoin
- ○ Intravenous lorazepam followed by long term oral valproate
- ○ Long term oral valproate

○ No antiepileptic drug therapy is indicated

● Intravenous lorazepam **Correct**

○ Intravenous phenytoin followed by long term oral phenytoin

○ Intravenous lorazepam followed by long term oral valproate

○ Long term oral valproate

Key Learning Points

Neurology, Therapeutics

- Consider alcohol withdrawal as the cause of seizures in a patient with heavy alcohol intake. Management is with benzodiazepines, not anti-epileptics.

Explanation

The patient is experiencing alcohol withdrawal seizures which typically occur seven to 48 hours after cessation of drinking.

If no underlying structural lesion is detected and the patient does not have an epileptic disorder, no long term antiepileptic drug therapy is indicated.

However, intravenous administration of lorazepam has been shown to reduce the risk of recurrent seizures.

The absolute risk reduction for recurrence within six hours is 21%.

Next Question

A 44-year-old female teacher collapsed in the classroom after complaining of a severe headache associated with nausea.

She had been well in the preceding few days. She had a past medical history of hypertension and diabetes and took regular atenolol and metformin. She did not smoke or drink.

On arrival in the Emergency department, her Glasgow coma scale was 12/15 (motor 6 vocal 3 eyes 3) and there was nuchal rigidity. Her blood pressure was 145/85 mmHg, pulse was 90 beats per minute and regular and temperature was 37.1°C. Heart sounds were normal and the chest appeared clear.

Cranial nerve examination revealed a left dilated unreactive pupil with oculoparesis of the left medial rectus. Fundoscopy was normal. There were no obvious focal neurological signs on examining the peripheral nervous system, although both plantar responses were extensor.

A lumbar puncture was performed. There was straw-coloured fluid and the following data was gathered:

Opening pressure	15 cmH_2O	(6-18)
CSF white cell count	6 cells per ml	(<5)
CSF red cell count	1450 cells per ml	(<5)
CSF protein	0.46 g/L	(0.15-0.45)
Cytospin	Negative for cells	

What is the likely diagnosis?

(Please select 1 option)

○ Posterior communicating artery aneurysm

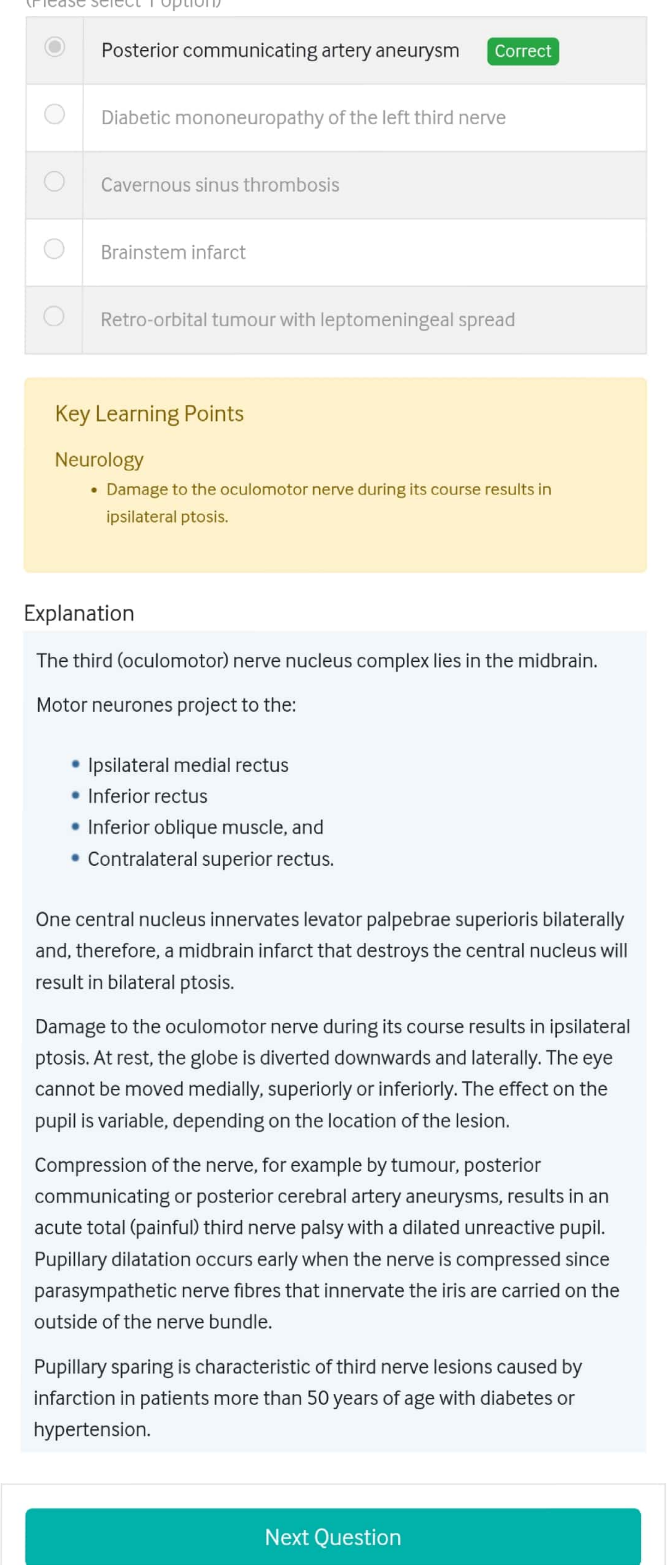
○ Diabetic mononeuropathy of the left third nerve

○ Cavernous sinus thrombosis

○ Brainstem infarct

○ Retro-orbital tumour with leptomeningeal spread

Submit Answer



(Please select 1 option)

● Posterior communicating artery aneurysm **Correct**

○ Diabetic mononeuropathy of the left third nerve

○ Cavernous sinus thrombosis

○ Brainstem infarct

○ Retro-orbital tumour with leptomeningeal spread

Key Learning Points

Neurology

- Damage to the oculomotor nerve during its course results in ipsilateral ptosis.

Explanation

The third (oculomotor) nerve nucleus complex lies in the midbrain.

Motor neurones project to the:

- Ipsilateral medial rectus
- Inferior rectus
- Inferior oblique muscle, and
- Contralateral superior rectus.

One central nucleus innervates levator palpebrae superioris bilaterally and, therefore, a midbrain infarct that destroys the central nucleus will result in bilateral ptosis.

Damage to the oculomotor nerve during its course results in ipsilateral ptosis. At rest, the globe is diverted downwards and laterally. The eye cannot be moved medially, superiorly or inferiorly. The effect on the pupil is variable, depending on the location of the lesion.

Compression of the nerve, for example by tumour, posterior communicating or posterior cerebral artery aneurysms, results in an acute total (painful) third nerve palsy with a dilated unreactive pupil. Pupillary dilatation occurs early when the nerve is compressed since parasympathetic nerve fibres that innervate the iris are carried on the outside of the nerve bundle.

Pupillary sparing is characteristic of third nerve lesions caused by infarction in patients more than 50 years of age with diabetes or hypertension.

A 24-year-old woman presents with an eight week history of headaches, which have deteriorated markedly over the last two days and have resulted in her admission.

These headaches are distressing to her; they have been problematical in the morning but she has found some relief from paracetamol. However, they are now constant and intolerable. The only other information is that she has gained 6 kg in weight the last six months.

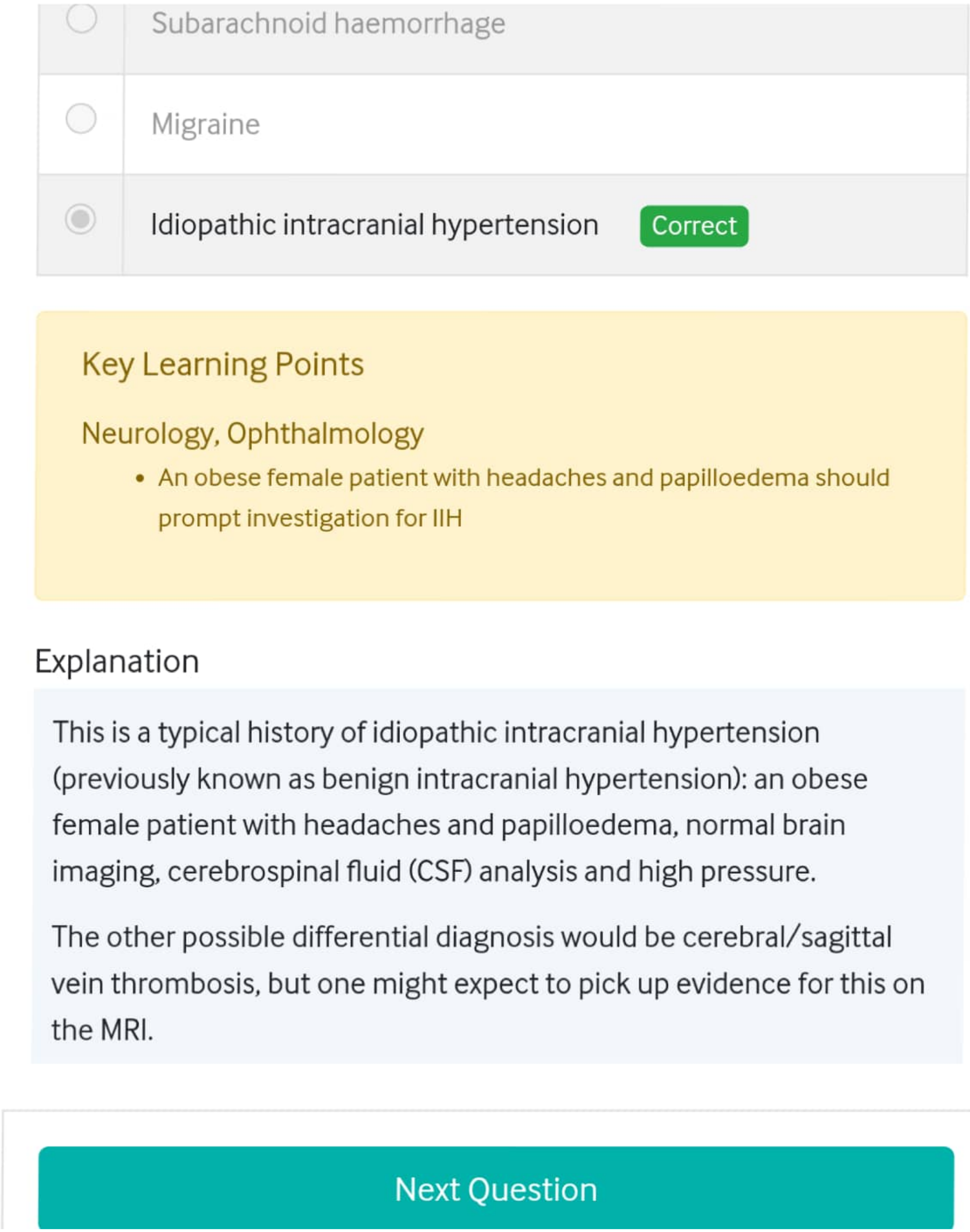
On examination she is noted to be obese with a BMI of 32 kg/m^2 and a blood pressure of 122/76 mmHg. Fundoscopy reveals bilateral swelling of both optic discs with loss of venous pulsation but otherwise neurological examination is normal. Investigations reveal normal MRI appearances of the brain and a lumbar puncture reveals an opening pressure of 30 cm H_2O but CSF analysis is normal.

What is the most likely diagnosis?

(Please select 1 option)

○	Multiple sclerosis
○	Tension type headache
○	Subarachnoid haemorrhage
○	Migraine
○	Idiopathic intracranial hypertension

Submit Answer



○ Subarachnoid haemorrhage

○ Migraine

● Idiopathic intracranial hypertension **Correct**

Key Learning Points

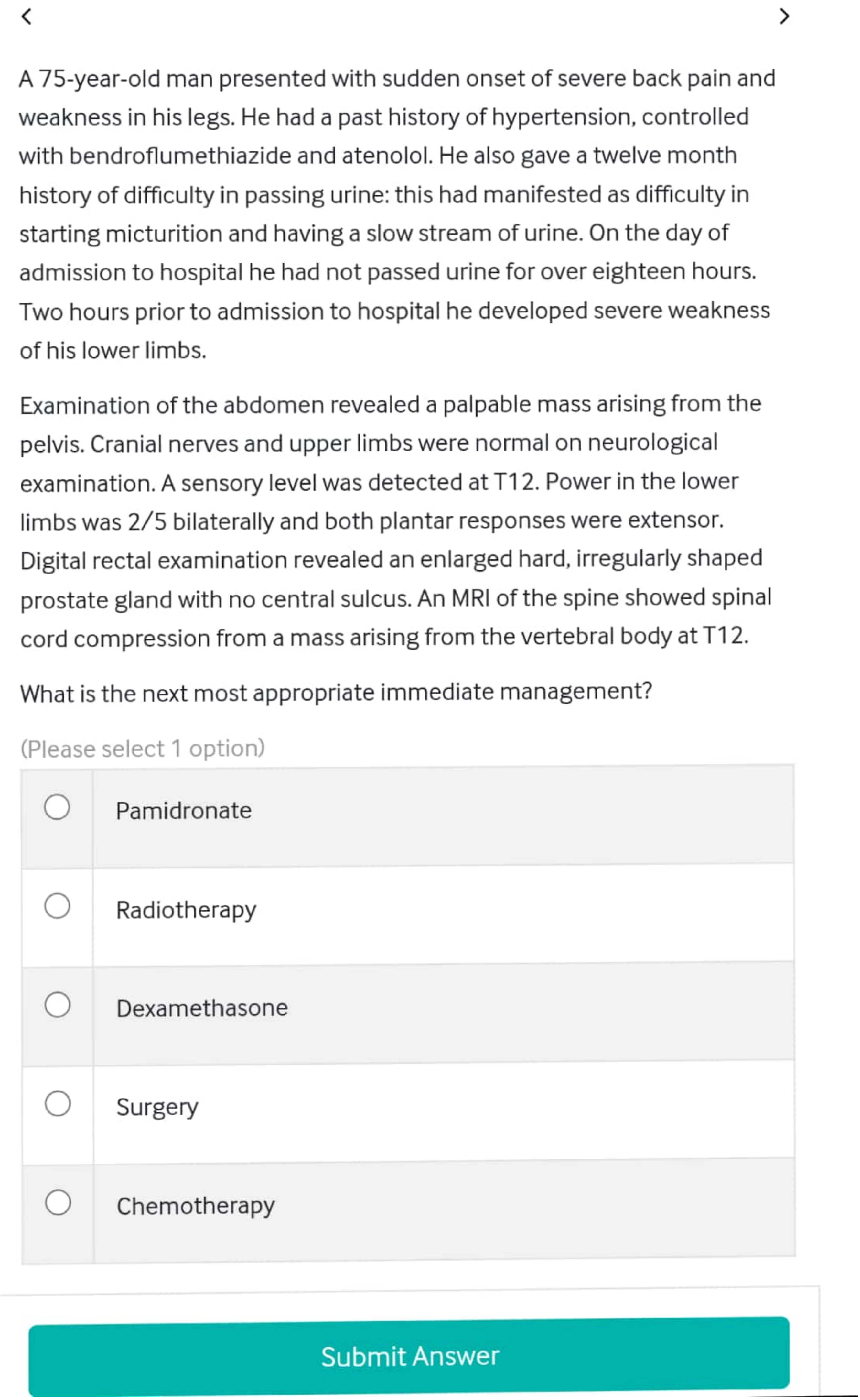
Neurology, Ophthalmology

- An obese female patient with headaches and papilloedema should prompt investigation for IIH

Explanation

This is a typical history of idiopathic intracranial hypertension (previously known as benign intracranial hypertension): an obese female patient with headaches and papilloedema, normal brain imaging, cerebrospinal fluid (CSF) analysis and high pressure.

The other possible differential diagnosis would be cerebral/sagittal vein thrombosis, but one might expect to pick up evidence for this on the MRI.



A 75-year-old man presented with sudden onset of severe back pain and weakness in his legs. He had a past history of hypertension, controlled with bendroflumethiazide and atenolol. He also gave a twelve month history of difficulty in passing urine: this had manifested as difficulty in starting micturition and having a slow stream of urine. On the day of admission to hospital he had not passed urine for over eighteen hours. Two hours prior to admission to hospital he developed severe weakness of his lower limbs.

Examination of the abdomen revealed a palpable mass arising from the pelvis. Cranial nerves and upper limbs were normal on neurological examination. A sensory level was detected at T12. Power in the lower limbs was 2/5 bilaterally and both plantar responses were extensor. Digital rectal examination revealed an enlarged hard, irregularly shaped prostate gland with no central sulcus. An MRI of the spine showed spinal cord compression from a mass arising from the vertebral body at T12.

What is the next most appropriate immediate management?

(Please select 1 option)

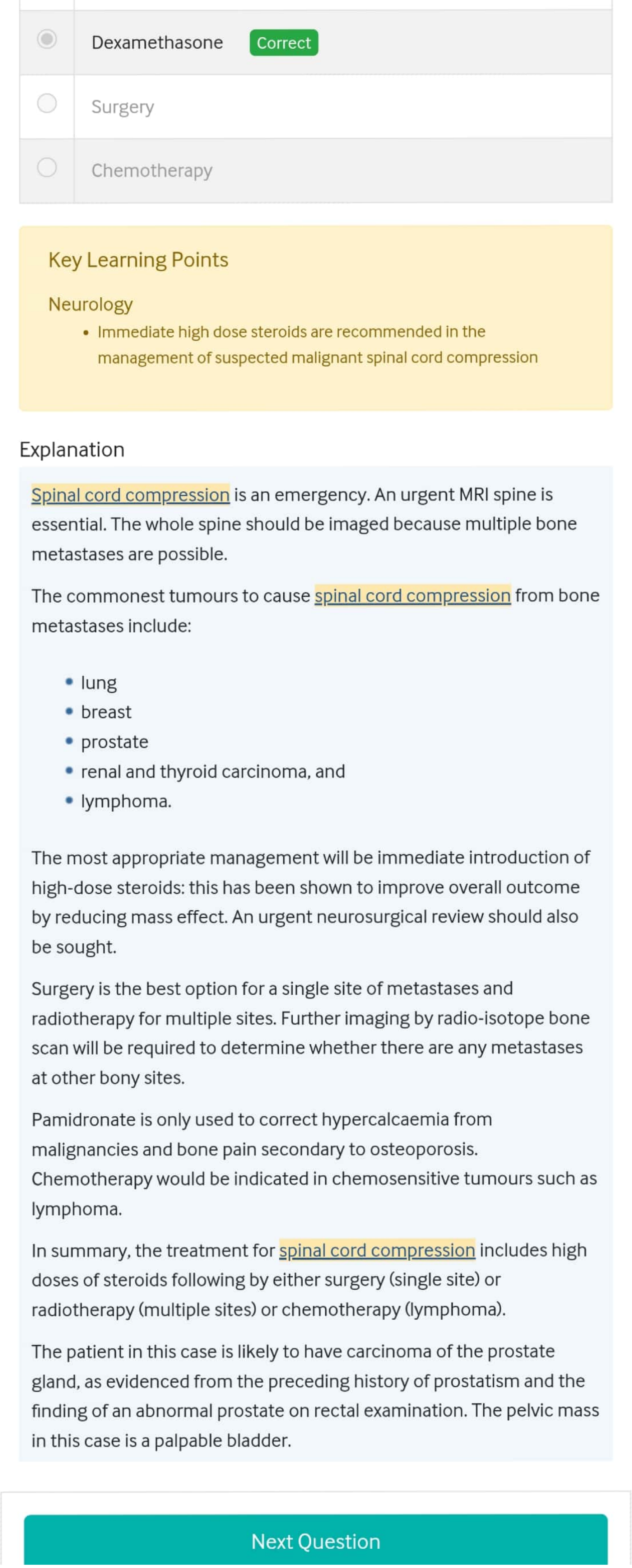
○ Pamidronate

○ Radiotherapy

○ Dexamethasone

○ Surgery

○ Chemotherapy



● Dexamethasone Correct

○ Surgery

○ Chemotherapy

Key Learning Points

Neurology

- Immediate high dose steroids are recommended in the management of suspected malignant spinal cord compression

Explanation

Spinal cord compression is an emergency. An urgent MRI spine is essential. The whole spine should be imaged because multiple bone metastases are possible.

The commonest tumours to cause spinal cord compression from bone metastases include:

- lung
- breast
- prostate
- renal and thyroid carcinoma, and
- lymphoma.

The most appropriate management will be immediate introduction of high-dose steroids: this has been shown to improve overall outcome by reducing mass effect. An urgent neurosurgical review should also be sought.

Surgery is the best option for a single site of metastases and radiotherapy for multiple sites. Further imaging by radio-isotope bone scan will be required to determine whether there are any metastases at other bony sites.

Pamidronate is only used to correct hypercalcaemia from malignancies and bone pain secondary to osteoporosis. Chemotherapy would be indicated in chemosensitive tumours such as lymphoma.

In summary, the treatment for spinal cord compression includes high doses of steroids following by either surgery (single site) or radiotherapy (multiple sites) or chemotherapy (lymphoma).

The patient in this case is likely to have carcinoma of the prostate gland, as evidenced from the preceding history of prostatism and the finding of an abnormal prostate on rectal examination. The pelvic mass in this case is a palpable bladder.

A 60-year-old man presented with a 12 month history of forgetfulness and paroxysmal drowsiness.

He was examined by the GP who found bilateral bradykinesia and rigidity in the upper limbs. He was started on L-dopa. Six weeks later he developed formed visual hallucinations. The visual hallucinations persisted despite stopping the L-dopa. The GP started him on a small dose of haloperidol (5 mg). However, the patient developed severe vomiting and drowsiness; he recovered when haloperidol was discontinued.

He had mild bradykinesias and rigidity in the upper limbs with rest tremor of the left arm. Gait was normal. His mini mental examination was 20/30. The remainder of the examination was normal.

Investigations showed:

Haemoglobin	165 g/L	(130-180)
White cell count	8.9×10^9/L	(4-11)
Platelets	290×10^9/L	(150-400)
Serum sodium	139 mmol/L	(137-144)
Serum potassium	3.8 mmol/L	(3.5-4.9)
Serum urea	6.7 mmol/L	(2.5-7.5)
Serum creatinine	91 µmol/L	(60-110)
Serum albumin	39 g/L	(37-49)
Serum total bilirubin	19 µmol/L	(1-22)
Serum aspartate aminotransferase	28 U/L	(5-35)
Serum alkaline phosphatase	99 U/L	(45-105)
Serum calcium (corrected)	2.4 mmol/L	(2.2-2.6)
Plasma glucose	4.2 mmol/L	(3.0-6.0)
Free T4	11 pmol/L	(10-22)
Free T3	8 pmol/L	(5-10)
Plasma TSH	3 mU/L	(0.4-5.0)
Serum vitamin B_{12}	228 ng/L	(160-760)
VDRL	Negative	-

CT brain and EEG were reported as being normal.

What is the most likely diagnosis?

(Please select 1 option)

○	Vascular dementia
○	Idiopathic Parkinson's disease
○	Schizophrenia
○	Diffuse Lewy body disease
○	Alzheimer's disease

Submit Answer

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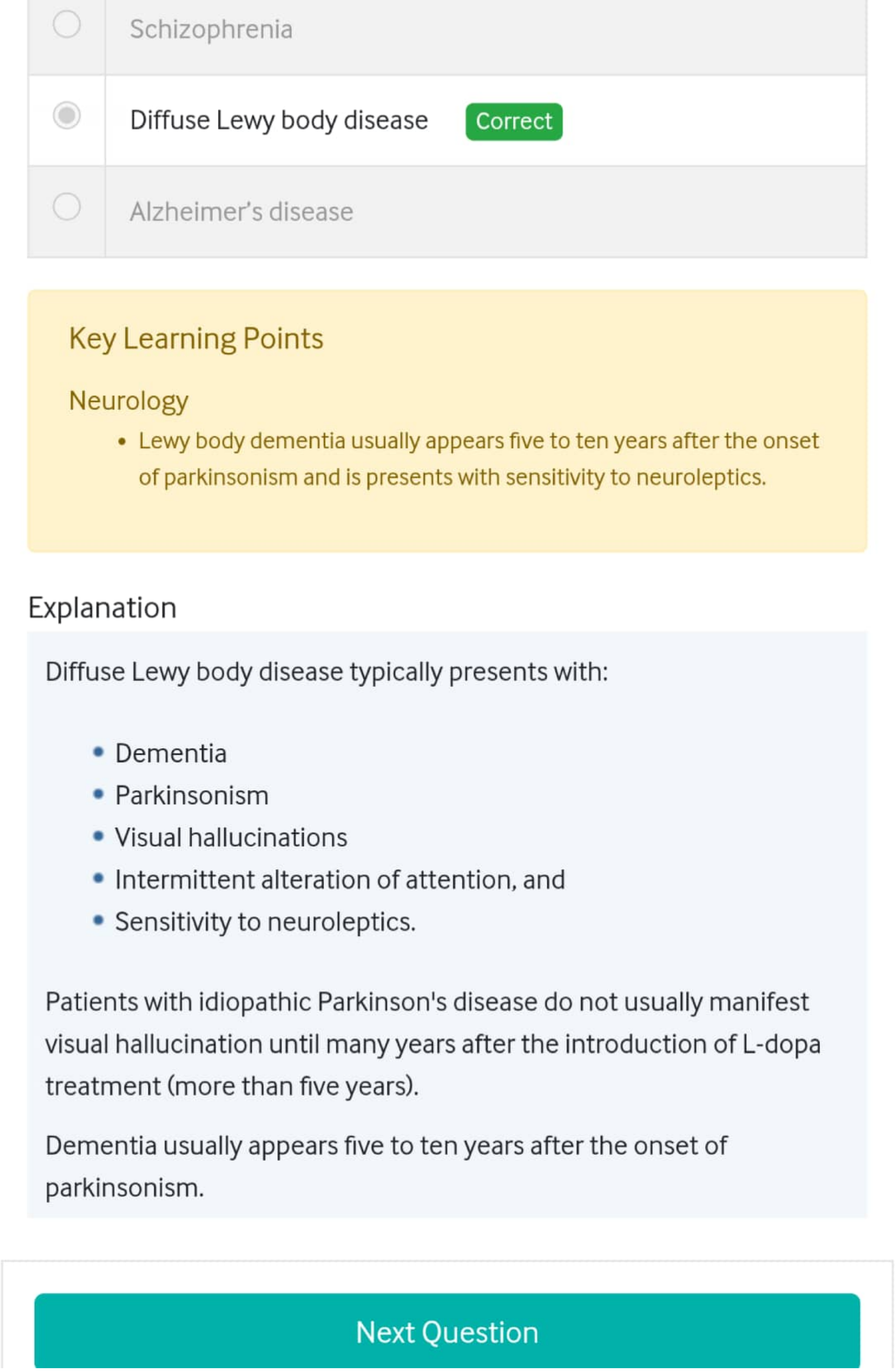


○ Schizophrenia

● Diffuse Lewy body disease

○ Alzheimer's disease

Key Learning Points

Neurology

- Lewy body dementia usually appears five to ten years after the onset of parkinsonism and is presents with sensitivity to neuroleptics.

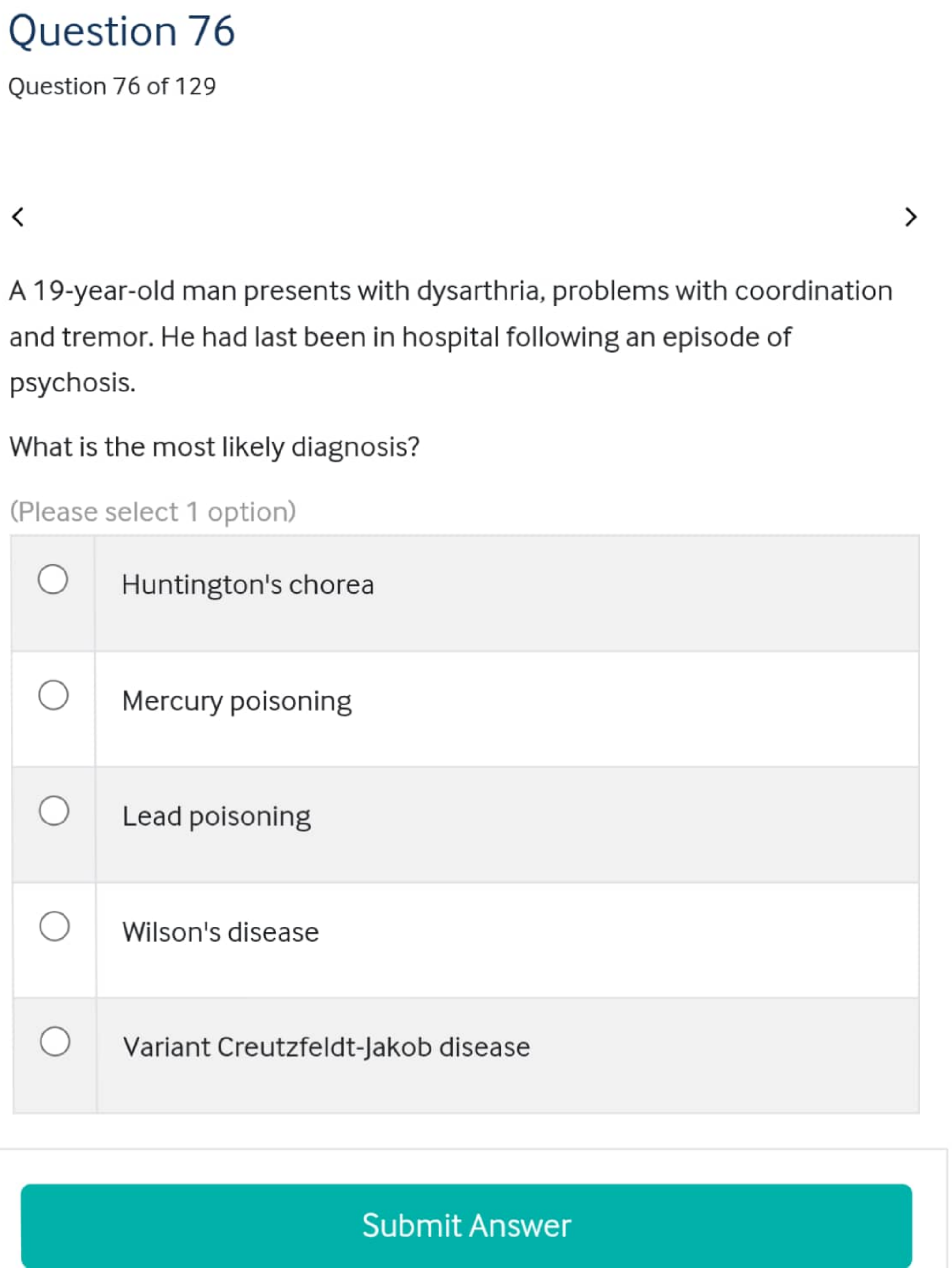
Explanation

Diffuse Lewy body disease typically presents with:

- Dementia
- Parkinsonism
- Visual hallucinations
- Intermittent alteration of attention, and
- Sensitivity to neuroleptics.

Patients with idiopathic Parkinson's disease do not usually manifest visual hallucination until many years after the introduction of L-dopa treatment (more than five years).

Dementia usually appears five to ten years after the onset of parkinsonism.



Question 76

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A 19-year-old man presents with dysarthria, problems with coordination and tremor. He had last been in hospital following an episode of psychosis.

What is the most likely diagnosis?

(Please select 1 option)

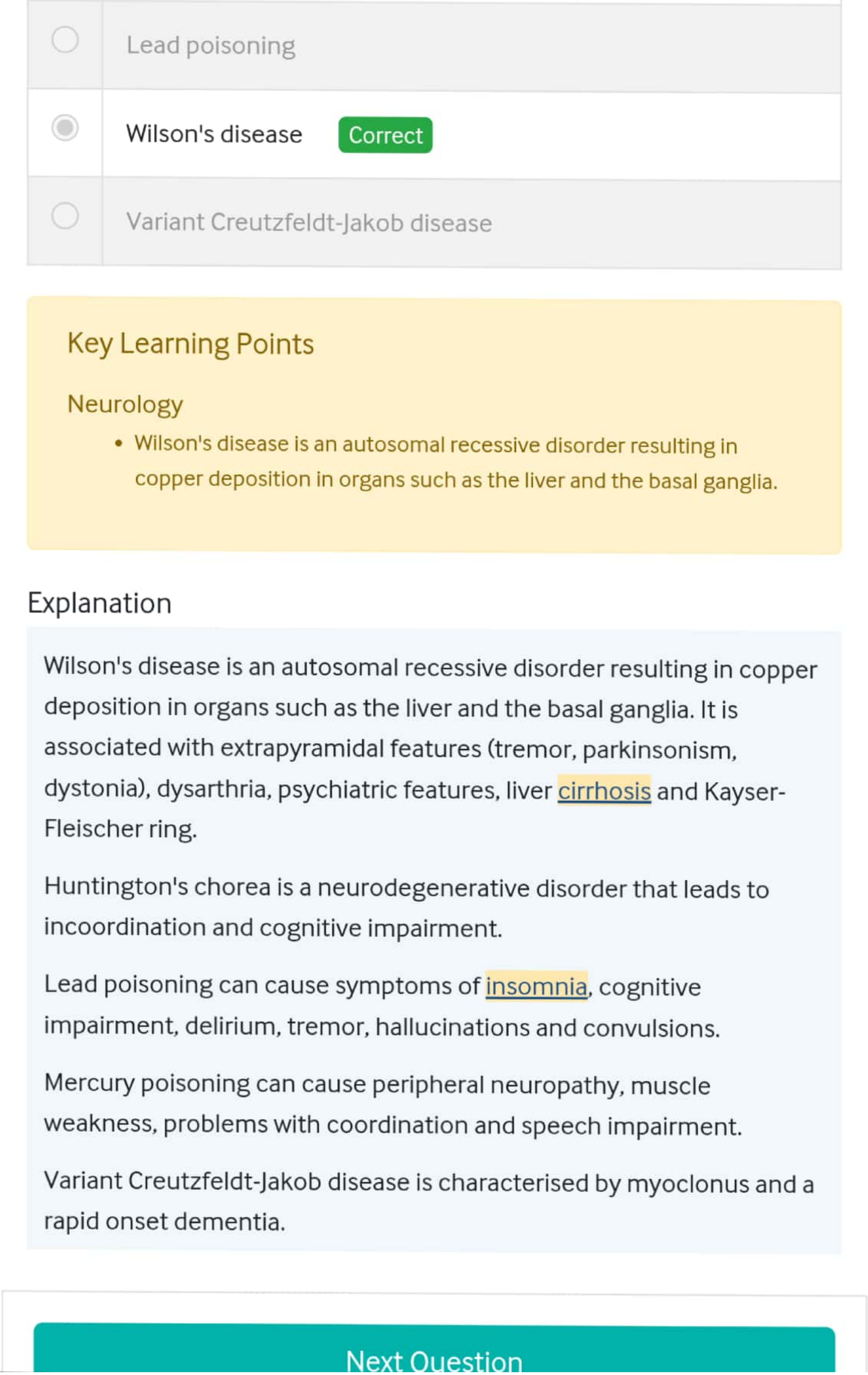
○ Huntington's chorea

○ Mercury poisoning

○ Lead poisoning

○ Wilson's disease

○ Variant Creutzfeldt-Jakob disease



○ Lead poisoning

● Wilson's disease Correct

○ Variant Creutzfeldt-Jakob disease

Key Learning Points

Neurology

- Wilson's disease is an autosomal recessive disorder resulting in copper deposition in organs such as the liver and the basal ganglia.

Explanation

Wilson's disease is an autosomal recessive disorder resulting in copper deposition in organs such as the liver and the basal ganglia. It is associated with extrapyramidal features (tremor, parkinsonism, dystonia), dysarthria, psychiatric features, liver cirrhosis and Kayser-Fleischer ring.

Huntington's chorea is a neurodegenerative disorder that leads to incoordination and cognitive impairment.

Lead poisoning can cause symptoms of insomnia, cognitive impairment, delirium, tremor, hallucinations and convulsions.

Mercury poisoning can cause peripheral neuropathy, muscle weakness, problems with coordination and speech impairment.

Variant Creutzfeldt-Jakob disease is characterised by myoclonus and a rapid onset dementia.

A 60-year old male presents with five days history of tingling of his fingers and toes.

Over the last two days he noticed progressive weakness of his upper and lower limbs. On admission he is unable to ambulate unaided. On examination he has a tetraparesis, areflexia with flexor plantar response.

CSF examination shows:

Red cells	0	-
White cells	0	-
CSF glucose	3.6 mmol/L	(3.3-4.4)
Plasma glucose	5.2 mmol/L	(3.0-6.0)
CSF protein	1.3 g/L	(0.15-0.45)

What is the most appropriate treatment?

(Please select 1 option)

○ Administration of IV immunoglobulin

○ No treatment required just physiotherapy

○ Pyridostigmine

○ Steroid therapy

○ L dopa

What is the most appropriate treatment?

(Please select 1 option)

● Administration of IV immunoglobulin **Correct**

○ No treatment required just physiotherapy

○ Pyridostigmine

○ Steroid therapy

○ L dopa

Key Learning Points

Neurology

- Intravenous immunoglobulins (0.4 g/kg for five days) and plasma exchange are the treatment of choice in Guillain-Barré.

Explanation

Intravenous immunoglobulins (0.4 g/kg for five days) and plasma exchange are the treatment of choice in Guillain-Barré.

They improve the long term prognosis and have the same efficacy.

Next Question

A 26-year-old woman presents to the Emergency department with a severe headache.

She reports that this has been getting steadily worse over a period of two to three months and is now also associated with blurred vision. She has a past history of depression: she attributes this and feeling of low self-esteem to her weight problem and bad skin; but for the past four months has tried to address these problems by joining Weight Watchers and starting treatment for her acne from her GP.

On examination she is overweight and has moderately severe acne. She is afebrile and there are no signs of nuchal rigidity. Oropharynx is benign. Neurological examination is normal except for blurred disc margins bilaterally and a limited ability to abduct the left eye.

Which investigation would best guide management?

(Please select 1 option)

○	CT scan sinuses
○	Cerebral angiography
○	Lumbar puncture
○	Overnight oxymetry (sleep study)
○	Visual Field Testing

Submit Answer

● Lumbar puncture **Correct**

○ Overnight oxymetry (sleep study)

○ Visual Field Testing

Key Learning Points

Neurology

- Idiopathic Intracranial Hypertension requires MR venogram and LP in order to confirm diagnosis

Explanation

This is a typical history and presentation of idiopathic intracranial hypertension (IIH). IIH typically affects young obese women.

Other risk factors include:

- The oral contraceptive pill
- Treatments for acne (tetracycline, nitrofurantoin, retinoids), and
- Hypervitaminosis A.

Presentation is with a headache, which may be severe. There may be loss of peripheral vision and impaired visual acuity if papilloedema is severe. Reduction in colour vision is common and a significant number of patients develop a CN VI palsy.

CT scan is often normal; the diagnosis is confirmed by finding an elevated cerebrospinal fluid (CSF) opening pressure (more than 20 cmH_2O). CSF protein, glucose and cell count will be normal.

The differential diagnosis includes venous sinus thrombosis; increased use of MRI has shown that small thromboses are commoner than previously thought in these patients.

MRI and/or MRI venography is essential in these patients.

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A 64-year-old man was seen on the acute medical intake, having presented with a four month history of weight loss and swallowing difficulties.

He had also noticed reduced dexterity of his left hand and fingers and struggled with writing and fine manipulation. He denied any bowel, bladder or breathing difficulties and had not noticed any obvious change in his appetite other than occasional episodes of nasal regurgitation.

He had a past medical history of osteoarthritis affecting his neck and hands and hypertension. He took regular diclofenac and bendroflumethiazide. He was a smoker of 25 cigarettes per day and drank 20 units of alcohol per week.

On examination, he had a bulbar dysarthria and there was generalised loss of muscle bulk.

On cranial nerve examination ocular movements were full with the only significant finding of tongue wasting and fasciculations.

Examination of the upper limb revealed wasting of the left dorsal interossei and thenar eminence together with fasciculations. Tone appeared increased in the left arm with brisk reflexes. Sensation and cerebellar function were intact. On examination of the lower limb, there was some evidence of fasciculations with increased tone, bilaterally brisk reflexes and extensor plantar responses. Chest, cardiovascular and abdominal examinations were normal.

An MRI scan of his cervical spine showed:
Degenerative joint disease affecting C3-4.

EMG/nerve conduction studies showed:
Fasciculations detected in proximal muscles and small muscles of the left arm.
Fasciculations detected in proximal muscles of the right arm.
Normal motor conduction and sensory studies.

Given the history and the clinical findings, what is the most likely diagnosis in this patient?

(Please select 1 option)

○ Motor neurone disease

○ Paraneoplastic neuropathy and cerebral metastases

○ Multifocal motor neuropathy

○ Chronic inflammatory demyelinating polyneuropathy (CIDP)

○ Cervical myelopathy at C3/C4

Submit Answer

● Motor neurone disease **Correct**

○ Paraneoplastic neuropathy and cerebral metastases

○ Multifocal motor neuropathy

○ Chronic inflammatory demyelinating polyneuropathy (CIDP)

○ Cervical myelopathy at C3/C4

Key Learning Points

Neurology

- MND produces and EMG with fasicualtions in clinically normal and abnormal muscles with preserved motor conduction and sensory studies.

Explanation

This patient has findings in keeping with motor neuron disease, which is a progressive neurodegenerative disease involving the corticospinal, corticobulbar tracts and anterior horn cell.

The EMG/nerve conduction studies support this diagnosis showing generalised fasciculations in clinically normal and abnormal muscles with preserved motor conduction and sensory studies.

A cervical myelopathy at the C3/C4 level would lead to upper motor neurone signs in the legs and arms.

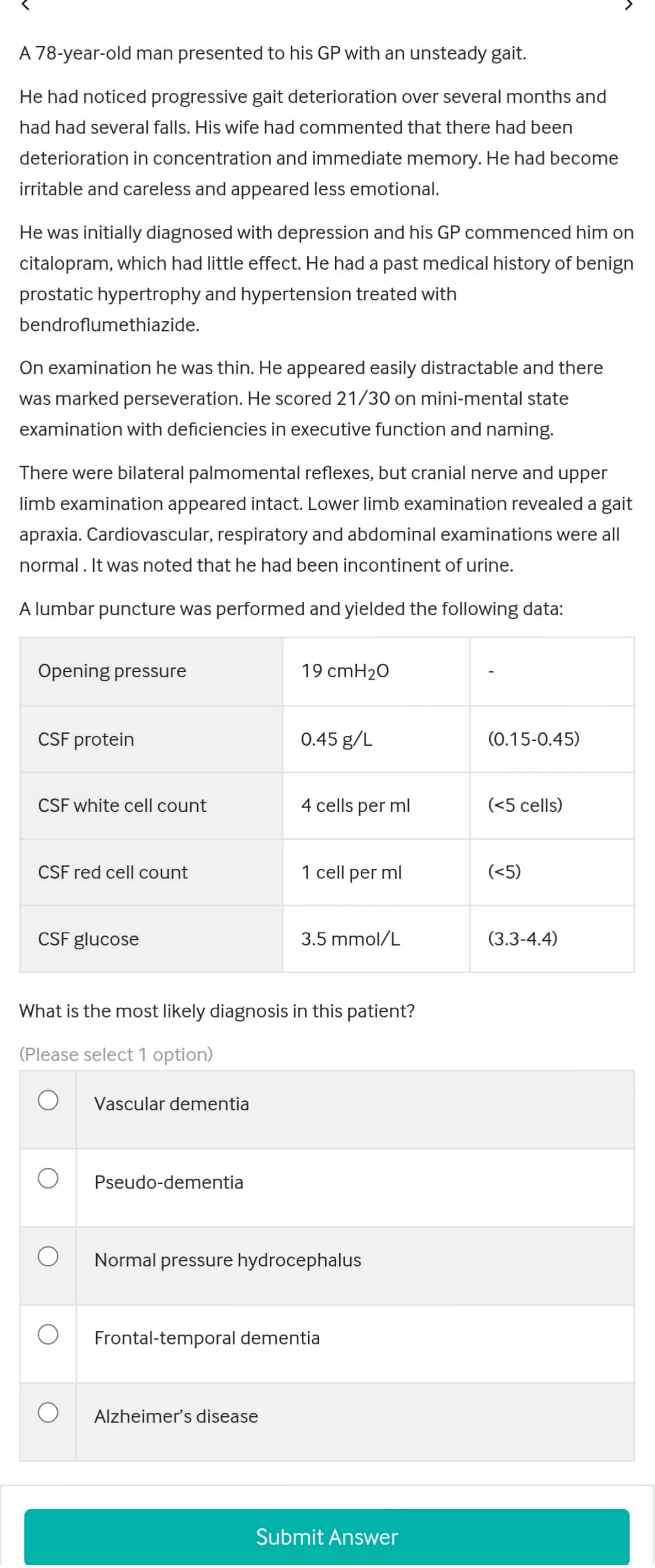
Chronic inflammatory demyelinating polyneuropathy (CIDP) is an inflammatory neuropathy associated with a preceding viral illness and would typically cause lower motor neurone signs and sensory disturbance.

Multi-focal motor neuropathy (MMN) is an important treatable differential diagnosis of motor neurone disease. MMN is an autoimmune neuropathy presenting with progressive weakness affecting an isolated limb. Fasciculations can occur in affected muscles, but cranial nerve involvement is rare.

The median survival time for motor neurone disease from onset of symptoms is three to five years. However, some patients survive a longer time (mainly primary lateral sclerosis).

Poor prognostic factors include low forced vital capacity (FVC) and older age.

Next Question



A 78-year-old man presented to his GP with an unsteady gait.

He had noticed progressive gait deterioration over several months and had had several falls. His wife had commented that there had been deterioration in concentration and immediate memory. He had become irritable and careless and appeared less emotional.

He was initially diagnosed with depression and his GP commenced him on citalopram, which had little effect. He had a past medical history of benign prostatic hypertrophy and hypertension treated with bendroflumethiazide.

On examination he was thin. He appeared easily distractable and there was marked perseveration. He scored 21/30 on mini-mental state examination with deficiencies in executive function and naming.

There were bilateral palmomental reflexes, but cranial nerve and upper limb examination appeared intact. Lower limb examination revealed a gait apraxia. Cardiovascular, respiratory and abdominal examinations were all normal . It was noted that he had been incontinent of urine.

A lumbar puncture was performed and yielded the following data:

Opening pressure	19 cmH_2O	-
CSF protein	0.45 g/L	(0.15-0.45)
CSF white cell count	4 cells per ml	(<5 cells)
CSF red cell count	1 cell per ml	(<5)
CSF glucose	3.5 mmol/L	(3.3-4.4)

What is the most likely diagnosis in this patient?

(Please select 1 option)

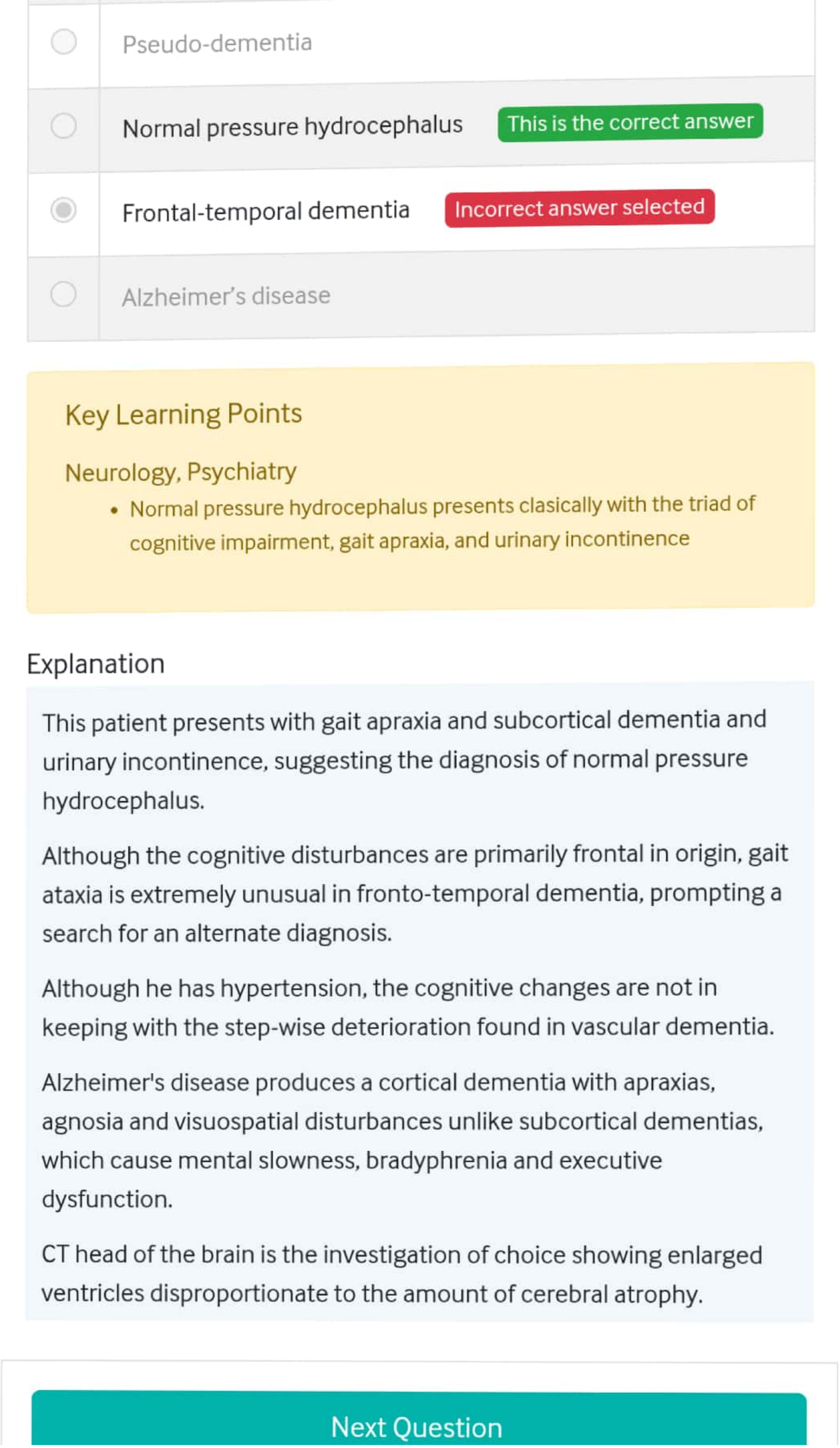
○ Vascular dementia

○ Pseudo-dementia

○ Normal pressure hydrocephalus

○ Frontal-temporal dementia

○ Alzheimer’s disease



○ Pseudo-dementia

○ Normal pressure hydrocephalus **This is the correct answer**

● Frontal-temporal dementia **Incorrect answer selected**

○ Alzheimer's disease

Key Learning Points

Neurology, Psychiatry

- Normal pressure hydrocephalus presents clasically with the triad of cognitive impairment, gait apraxia, and urinary incontinence

Explanation

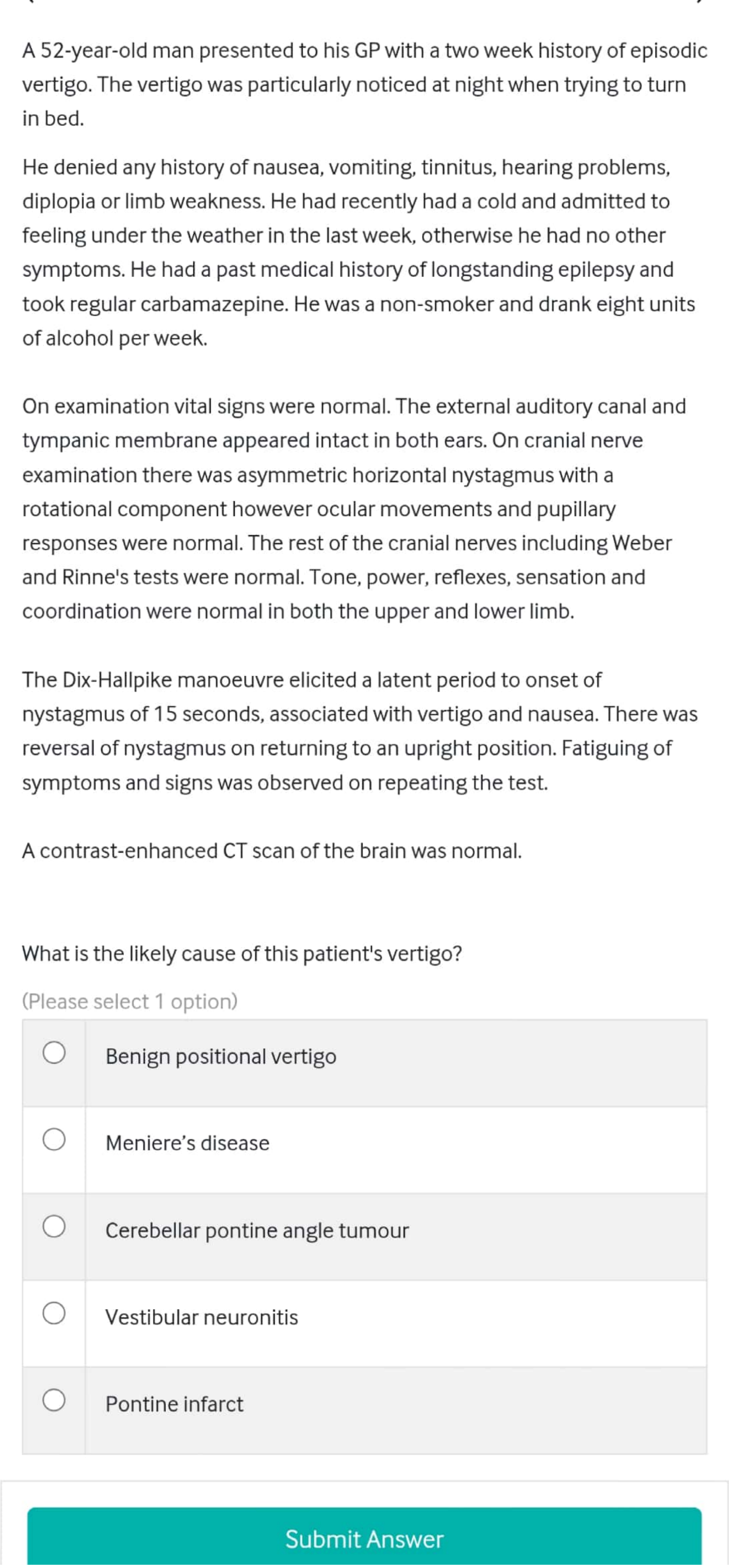
This patient presents with gait apraxia and subcortical dementia and urinary incontinence, suggesting the diagnosis of normal pressure hydrocephalus.

Although the cognitive disturbances are primarily frontal in origin, gait ataxia is extremely unusual in fronto-temporal dementia, prompting a search for an alternate diagnosis.

Although he has hypertension, the cognitive changes are not in keeping with the step-wise deterioration found in vascular dementia.

Alzheimer's disease produces a cortical dementia with apraxias, agnosia and visuospatial disturbances unlike subcortical dementias, which cause mental slowness, bradyphrenia and executive dysfunction.

CT head of the brain is the investigation of choice showing enlarged ventricles disproportionate to the amount of cerebral atrophy.



A 52-year-old man presented to his GP with a two week history of episodic vertigo. The vertigo was particularly noticed at night when trying to turn in bed.

He denied any history of nausea, vomiting, tinnitus, hearing problems, diplopia or limb weakness. He had recently had a cold and admitted to feeling under the weather in the last week, otherwise he had no other symptoms. He had a past medical history of longstanding epilepsy and took regular carbamazepine. He was a non-smoker and drank eight units of alcohol per week.

On examination vital signs were normal. The external auditory canal and tympanic membrane appeared intact in both ears. On cranial nerve examination there was asymmetric horizontal nystagmus with a rotational component however ocular movements and pupillary responses were normal. The rest of the cranial nerves including Weber and Rinne's tests were normal. Tone, power, reflexes, sensation and coordination were normal in both the upper and lower limb.

The Dix-Hallpike manoeuvre elicited a latent period to onset of nystagmus of 15 seconds, associated with vertigo and nausea. There was reversal of nystagmus on returning to an upright position. Fatiguing of symptoms and signs was observed on repeating the test.

A contrast-enhanced CT scan of the brain was normal.

What is the likely cause of this patient's vertigo?

(Please select 1 option)

○	Benign positional vertigo
○	Meniere’s disease
○	Cerebellar pontine angle tumour
○	Vestibular neuronitis
○	Pontine infarct

(Please select 1 option)

● Benign positional vertigo **Correct**

○ Meniere’s disease

○ Cerebellar pontine angle tumour

○ Vestibular neuronitis

○ Pontine infarct

Key Learning Points

Neurology

- Dix-Hallpike manoeuvre is a helpful bedside test in eliciting vertigo in benign positional vertigo.

Explanation

This patient gives a history of episodic vertigo, which appears positional, and is not associated with any brainstem features such as diplopia, facial weakness or long-tract signs and symptoms. In addition there is no history of tinnitus, hearing loss or headache.

The history and normal neurological examination would be in keeping with benign positional vertigo.

Dix-Hallpike manoeuvre is a helpful bedside test in eliciting vertigo and delayed torsional nystagmus with the fast phase beating downwards. On resuming the upright posture it is noted that the nystagmus has changed direction and after several repeated manoeuvres the nystagmus is more difficult to elicit.

Vestibular neuronitis is frequently associated with an antecedent upper respiratory tract infection and presents as abrupt onset vertigo, which is severe associated with nausea and vomiting lasting several days. There may be auditory eighth nerve abnormalities, and sensorineural deafness.

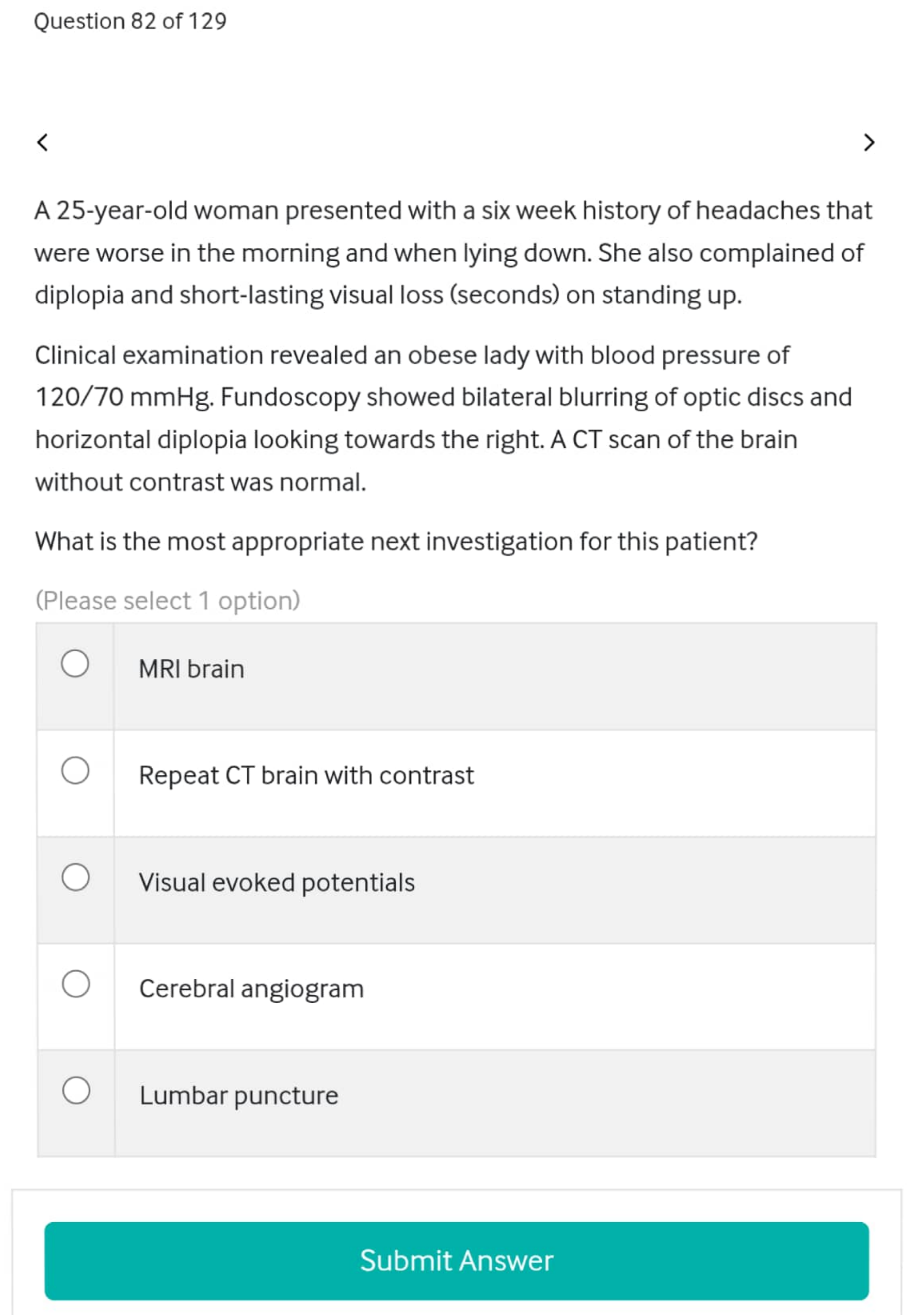
Meniere's disease is characterised by recurrent attacks of severe vertigo, vomiting, tinnitus and hearing loss.

The absence of abnormal neurological findings would make a cerebellar pontine angle tumour or a pontine infarct highly unlikely.

A cerebellar pontine angle tumour would present with ipsilateral cerebellar dysfunction and cranial nerve palsies (Vth, VIth, VIIth, VIIIth).

A pontine infarct would lead to similar cranial nerve dysfunction and long tract signs.

Next Question



A 25-year-old woman presented with a six week history of headaches that were worse in the morning and when lying down. She also complained of diplopia and short-lasting visual loss (seconds) on standing up.

Clinical examination revealed an obese lady with blood pressure of 120/70 mmHg. Fundoscopy showed bilateral blurring of optic discs and horizontal diplopia looking towards the right. A CT scan of the brain without contrast was normal.

What is the most appropriate next investigation for this patient?

(Please select 1 option)

○ MRI brain

○ Repeat CT brain with contrast

○ Visual evoked potentials

○ Cerebral angiogram

○ Lumbar puncture

○ Cerebral angiogram

● Lumbar puncture Correct

Key Learning Points

Neurology

- A lumbar puncture is needed to confirm high intracranial pressure (greater than 25 cm of H2O) in the diagnosis of IIH as well as normal CSF analysis.

Explanation

The most likely diagnosis is idiopathic intracranial hypertension.

It is associated with:

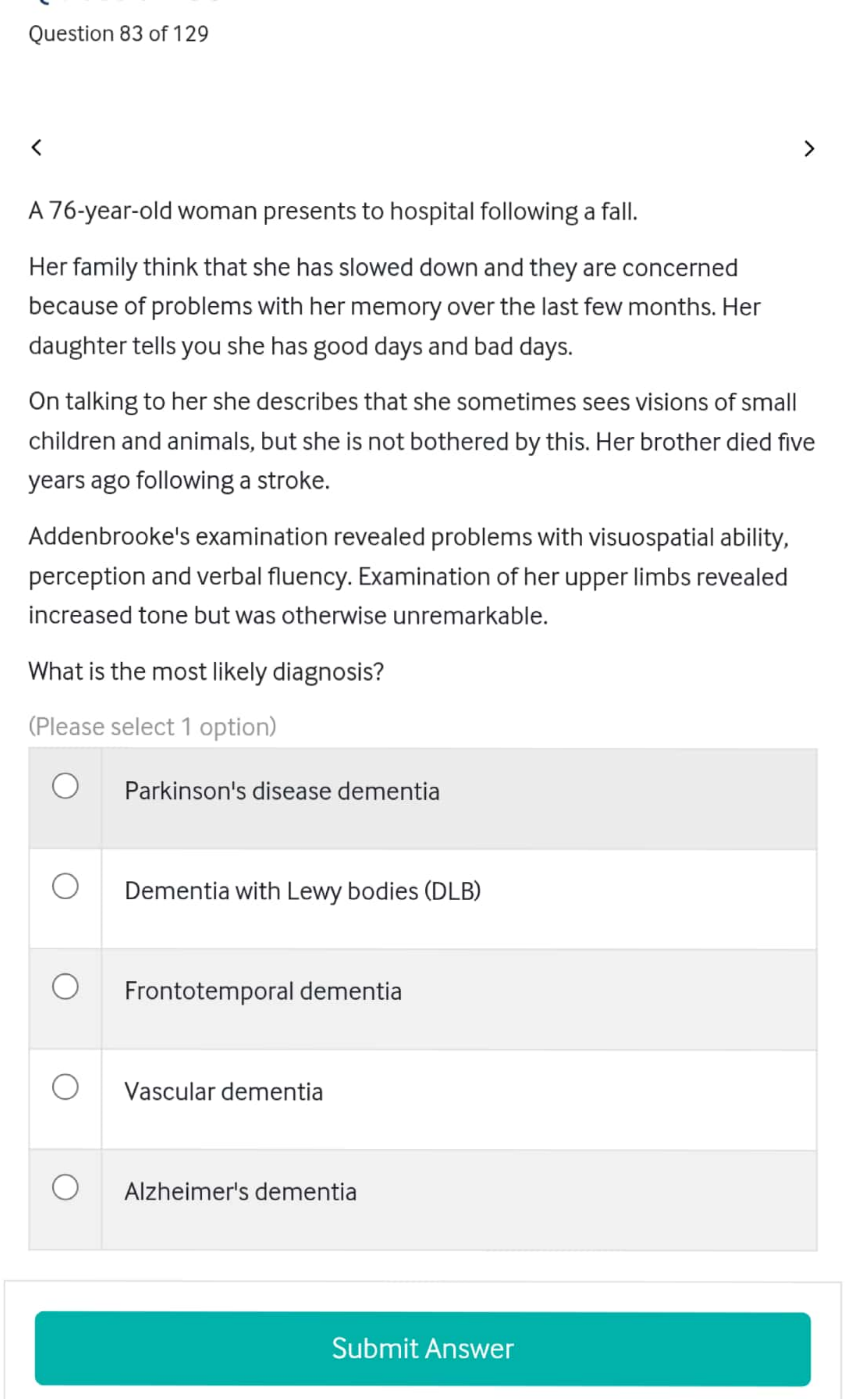
- Obesity
- Female sex
- Bilateral papilloedema
- Transient visual obscurations
- Horizontal diplopia, and
- Headaches (worst in the morning and lying down).

CT brain is needed to exclude a space occupying lesion and obstructing hydrocephalus.

A lumbar puncture is needed to confirm high intracranial pressure (greater than 25 cm of H_2O) and normal cerebrospinal fluid analysis.

MRI venogram is recommended afterwards to exclude cerebral sinus thrombosis.

Next Question



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A 76-year-old woman presents to hospital following a fall.

Her family think that she has slowed down and they are concerned because of problems with her memory over the last few months. Her daughter tells you she has good days and bad days.

On talking to her she describes that she sometimes sees visions of small children and animals, but she is not bothered by this. Her brother died five years ago following a stroke.

Addenbrooke's examination revealed problems with visuospatial ability, perception and verbal fluency. Examination of her upper limbs revealed increased tone but was otherwise unremarkable.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Parkinson's disease dementia
- ○ Dementia with Lewy bodies (DLB)
- ○ Frontotemporal dementia
- ○ Vascular dementia
- ○ Alzheimer's dementia

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○ Parkinson's disease dementia

● Dementia with Lewy bodies (DLB) Correct

○ Frontotemporal dementia

○ Vascular dementia

○ Alzheimer's dementia

Key Learning Points

Neurology

- Characteristic features of dementia with Lewy bodies are fluctuation of awareness from day-to-day, signs of parkinsonism and visual hallucinations or delusions occur frequently. Falls are also common. They have pronounced visuoperceptual impairment.

Explanation

Characteristic features of dementia with Lewy bodies are fluctuation of awareness from day-to-day, signs of parkinsonism and visual hallucinations or delusions occur frequently. Falls are also common. They have pronounced visuoperceptual impairment.

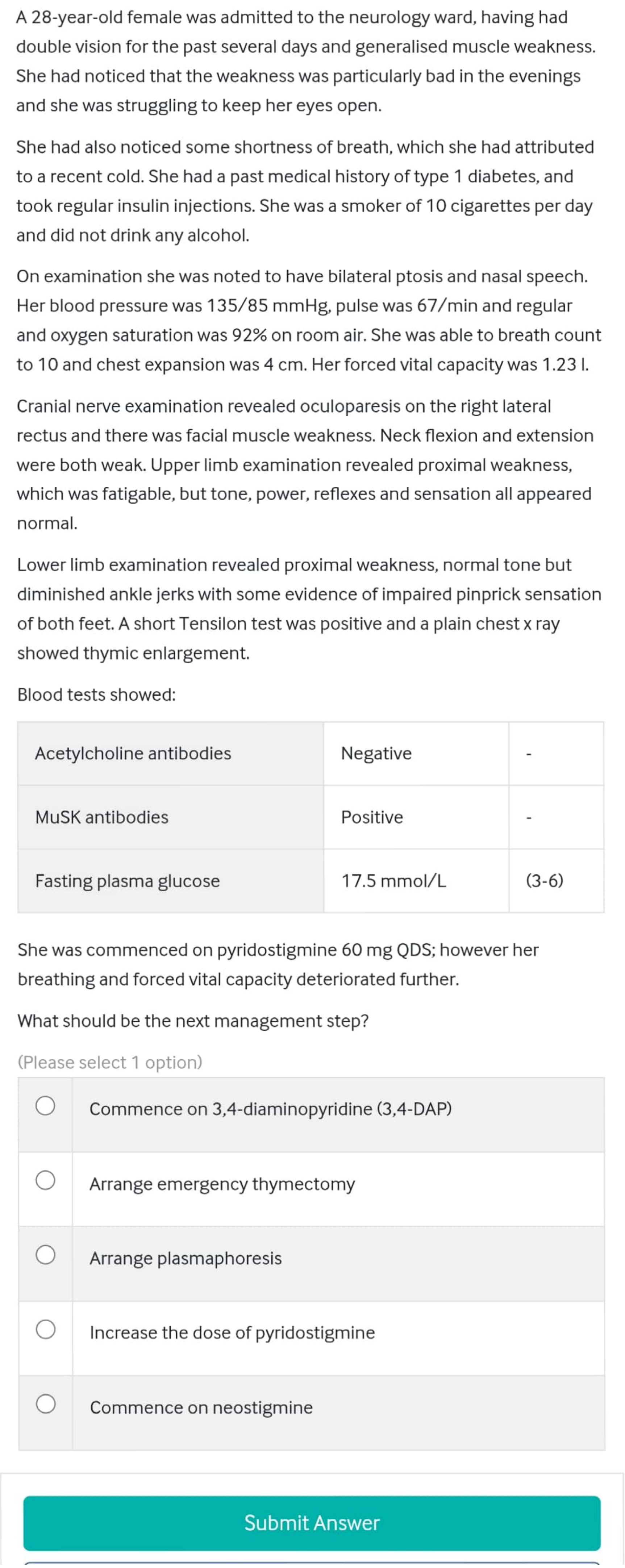
Alzheimer's disease is the most common cause of dementia and presents with memory problems, difficulties with activities of daily living (ADLs) and behavioural changes.

Frontotemporal dementia is more common under the age of 65. It presents with changes in behaviour and loss of insight more commonly than memory problems.

Parkinson's disease dementia has a similar pathological process to DLB and both are associated with progressive cognitive decline and parkinsonism. The dementia associated with Parkinson's disease occurs more than a year after the development of the motor symptoms of the disease.

Vascular dementia classically presents with periods of sudden decline and then stability with a stepwise progression. Patients have a history of stroke, hypertension or ischaemic heart disease.

Next Question



A 28-year-old female was admitted to the neurology ward, having had double vision for the past several days and generalised muscle weakness. She had noticed that the weakness was particularly bad in the evenings and she was struggling to keep her eyes open.

She had also noticed some shortness of breath, which she had attributed to a recent cold. She had a past medical history of type 1 diabetes, and took regular insulin injections. She was a smoker of 10 cigarettes per day and did not drink any alcohol.

On examination she was noted to have bilateral ptosis and nasal speech. Her blood pressure was 135/85 mmHg, pulse was 67/min and regular and oxygen saturation was 92% on room air. She was able to breath count to 10 and chest expansion was 4 cm. Her forced vital capacity was 1.23 l.

Cranial nerve examination revealed oculoparesis on the right lateral rectus and there was facial muscle weakness. Neck flexion and extension were both weak. Upper limb examination revealed proximal weakness, which was fatigable, but tone, power, reflexes and sensation all appeared normal.

Lower limb examination revealed proximal weakness, normal tone but diminished ankle jerks with some evidence of impaired pinprick sensation of both feet. A short Tensilon test was positive and a plain chest x ray showed thymic enlargement.

Blood tests showed:

Acetylcholine antibodies	Negative	-
MuSK antibodies	Positive	-
Fasting plasma glucose	17.5 mmol/L	(3-6)

She was commenced on pyridostigmine 60 mg QDS; however her breathing and forced vital capacity deteriorated further.

What should be the next management step?

(Please select 1 option)

○ Commence on 3,4-diaminopyridine (3,4-DAP)

○ Arrange emergency thymectomy

○ Arrange plasmaphoresis

○ Increase the dose of pyridostigmine

○ Commence on neostigmine

● Arrange plasmaphoresis **Correct**

○ Increase the dose of pyridostigmine

○ Commence on neostigmine

Key Learning Points

Neurology

- Myasthenic crisis requires plasma phoresis and transfer to ITU.

Explanation

The diagnosis is a myasthenic crisis.

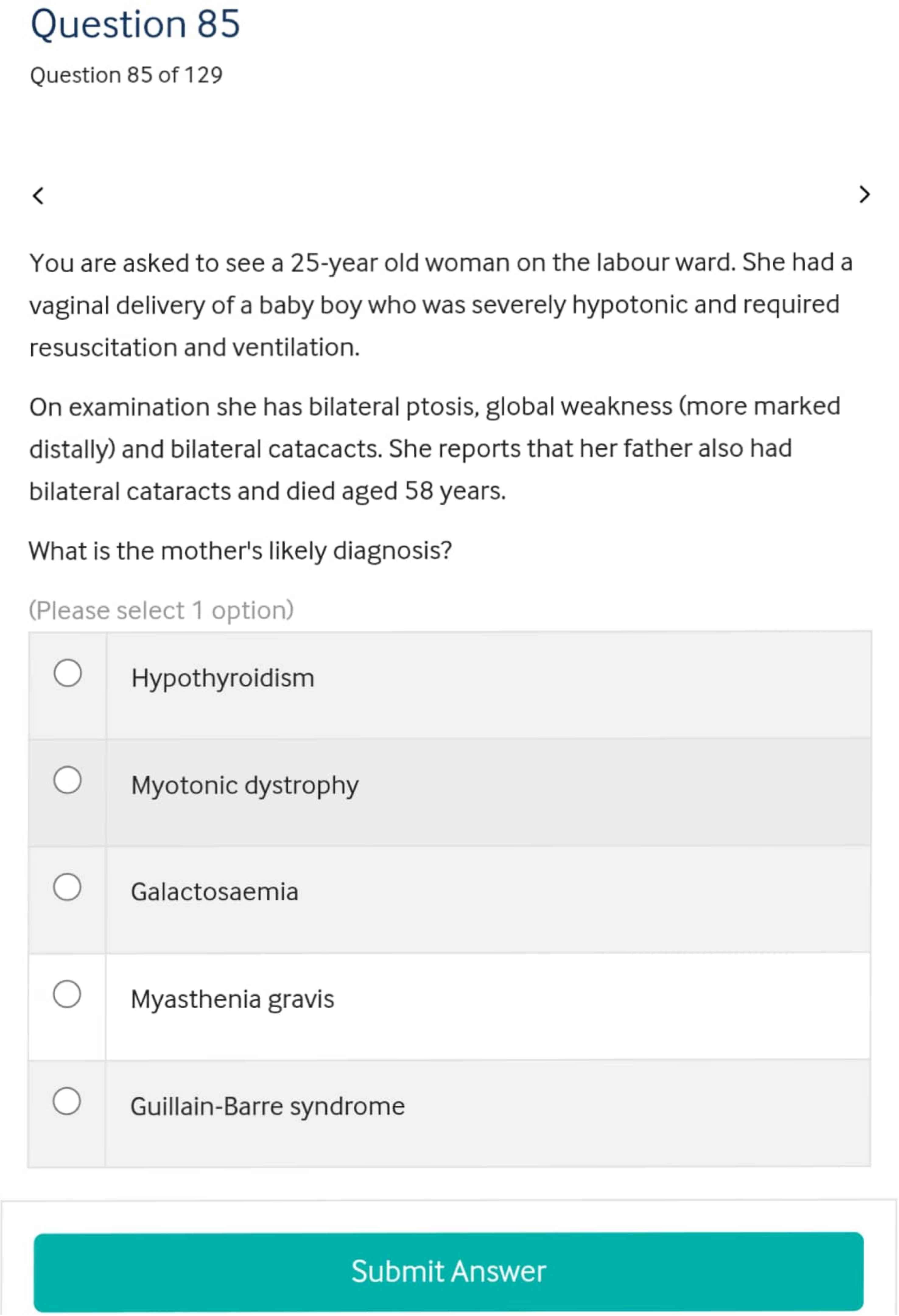
The patient has marked respiratory weakness with reduced breath count, reduced oxygen saturation, chest expansion and forced vital capacity. The treatment of choice is intravenous immunoglobulin or plasma exchange and transfer to intensive care.

This patient does have thymic enlargement and will subsequently require thymectomy. Given the poor respiratory capacity, however, she requires stabilisation first.

3,4-DAP is a treatment used in Eaton- Lambert syndrome, which is not the diagnosis in this case.

Prednisolone, azathioprine and cyclosporin are treatments used in myasthenia gravis, but their action is slow and with steroids there can be deterioration in the patient's condition before an improvement is seen.

Next Question



Question 85

Question 85 of 129

You are asked to see a 25-year old woman on the labour ward. She had a vaginal delivery of a baby boy who was severely hypotonic and required resuscitation and ventilation.

On examination she has bilateral ptosis, global weakness (more marked distally) and bilateral catacacts. She reports that her father also had bilateral cataracts and died aged 58 years.

What is the mother's likely diagnosis?

(Please select 1 option)

- ○ Hypothyroidism
- ○ Myotonic dystrophy
- ○ Galactosaemia
- ○ Myasthenia gravis
- ○ Guillain-Barre syndrome

● Myotonic dystrophy **Correct**

○ Galactosaemia

○ Myasthenia gravis

○ Guillain-Barre syndrome

Key Learning Points

Genetics, Neurology

- Myotonic dystrophy presents with distal weakness more so than proximal weakness as well as myotonia at an early age.

Explanation

Myotonic dystrophy (Steinert's disease) is the commonest adult muscular dystrophy.

Myotonia is often evident at an early age, but the severity is variable and many patients do not present until adulthood.

Distal weakness is often more marked than proximal weakness; there is a typical facial appearance due to atrophy of temporalis, masseter and facial muscles. Men often have frontal balding and some women also have hair loss. Cataracts are common.

Congenital myotonic dystrophy is a more severe form of the disease that occurs in children born to mothers who have established myotonic dystrophy. Babies are hypotonic and frequently require assisted ventilation.

Galactosaemia seldom presents with problems at birth. Babies develop diarrhoea and fail to gain weight. Liver disease ensues unless diagnosed. Cataracts are common.

Myasthenia gravis frequently presents in the third or fourth decade of life and exacerbations are common in pregnancy. While this requires special attention for the mother, the baby is unaffected.

Similarly, Guillain-Barre syndrome would not affect the baby.

Next Question

A 19-year-old right-handed man presented to hospital after suddenly developing right-sided weakness and difficulty speaking. There was no past medical history of note.

He was in university studying sociology and had recently returned from a summer holiday travelling with Christian Aid. He was a non-smoker and drank approximately eight units of alcohol per week.

On examination, he was afebrile. His pulse was 78 per minute and regular with a blood pressure of 130/75 mmHg. Auscultation of the heart revealed wide fixed splitting of the second heart sound and a soft systolic murmur in the second left intercostal space. The chest was clear on auscultation.

Neurological examination revealed an expressive and receptive dysphasia. Examination of the cranial nerves did not reveal any deficit. There was marked weakness of the right arm and leg with increased tone. Reflexes were brisk on the left, but hyperreflexic on the right with an extensor right plantar response. The left lower leg appeared erythematous and swollen.

Investigations revealed:

Haemoglobin	142 g/L	(130-180)
White cell count	8.5 $\times 10^9$/L	(4-11)
Platelets	399 $\times 10^9$/L	(150-400)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	3.9 mmol/L	(3.5-4.9)
Serum urea	4.7 mmol/L	(2.5-7.5)
Serum creatinine	95 μmol/L	(60-110)
Anti dsDNA	Negative	-
ANA	Negative	-
VDRL	Negative	-
Lupus anticoagulant	Negative	-
Protein C	100 U/dL	(80-125)
Protein S	110 U/dL	(80-120)
Antithrombin III	85 U/dL	(80-120)
D-dimer	12 mg/L	(<0.5)

The ECG showed right ventricular hypertrophy, right bundle branch block and partial right axis deviation.

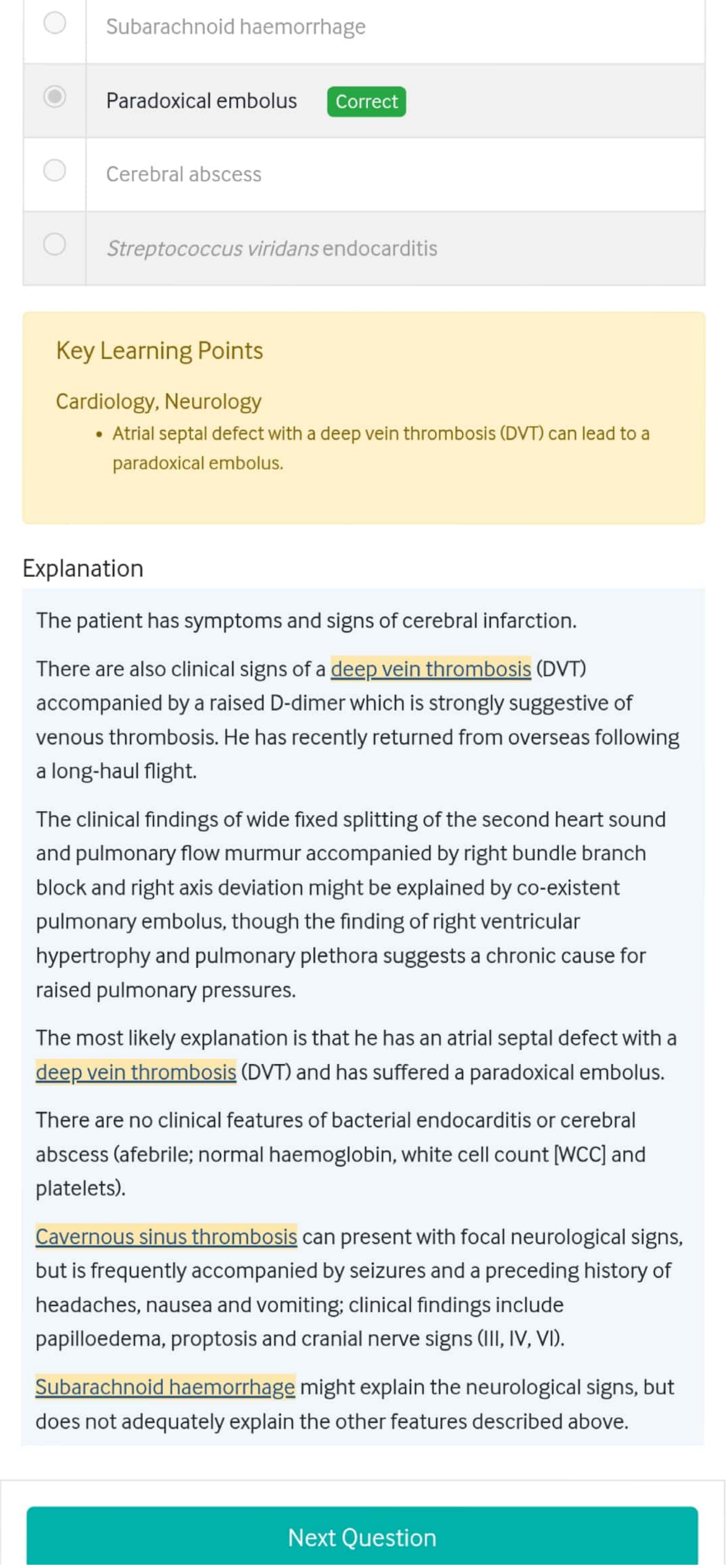
The chest x ray showed prominent pulmonary arteries and pulmonary plethora.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Cavernous sinus thrombosis
- ○ Subarachnoid haemorrhage
- ○ Paradoxical embolus
- ○ Cerebral abscess
- ○ *Streptococcus viridans* endocarditis

Submit Answer



○ Subarachnoid haemorrhage

● Paradoxical embolus

○ Cerebral abscess

○ *Streptococcus viridans* endocarditis

Key Learning Points

Cardiology, Neurology

- Atrial septal defect with a deep vein thrombosis (DVT) can lead to a paradoxical embolus.

Explanation

The patient has symptoms and signs of cerebral infarction.

There are also clinical signs of a deep vein thrombosis (DVT) accompanied by a raised D-dimer which is strongly suggestive of venous thrombosis. He has recently returned from overseas following a long-haul flight.

The clinical findings of wide fixed splitting of the second heart sound and pulmonary flow murmur accompanied by right bundle branch block and right axis deviation might be explained by co-existent pulmonary embolus, though the finding of right ventricular hypertrophy and pulmonary plethora suggests a chronic cause for raised pulmonary pressures.

The most likely explanation is that he has an atrial septal defect with a deep vein thrombosis (DVT) and has suffered a paradoxical embolus.

There are no clinical features of bacterial endocarditis or cerebral abscess (afebrile; normal haemoglobin, white cell count [WCC] and platelets).

Cavernous sinus thrombosis can present with focal neurological signs, but is frequently accompanied by seizures and a preceding history of headaches, nausea and vomiting; clinical findings include papilloedema, proptosis and cranial nerve signs (III, IV, VI).

Subarachnoid haemorrhage might explain the neurological signs, but does not adequately explain the other features described above.

Question 87

Question 87 of 129

‹ ›

A 47-year-old man presented to the Emergency department following sudden onset of left posterior auricular pain while in his garage working under his car. Within the next few hours he had developed vertigo and nausea and intractable hiccups.

He had a past medical history of migraine and hypertension and was taking atenolol 50 mg for both. He had no family history of medical or neurological problems.

On examination his blood pressure was 147/88 mmHg, pulse was 89/min and regular and heart sounds were normal. Flexion of the neck provoked pain and there was a degree of nuchal stiffness.

On cranial nerve examination he was dysarthric and had evidence of a left Horner's syndrome. Fundoscopy was normal. On testing ocular movements there was restriction of horizontal gaze. There was reduced pinprick sensation over the left side of the face. On upper limb examination there was reduced pinprick and temperature sensation over the right arm. On lower limb examination there also appeared to be reduced pinprick sensation over the right leg and he appeared to walk with an ataxic gait.

Investigations revealed:

Haemoglobin	135 g/L	(130-180)
White cell count	10.0 ×10^9/L	(4-11)
Platelets	345 ×10^9/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.8 mmol/L	(3.5-4.9)
Serum urea	4.7 mmol/L	(2.5-7.5)
Serum creatinine	88 μmol/L	(60-110)
Serum C reactive protein	<5 U/L	(<5)
Plasma glucose	4.3 mmol/L	(3-6)
Serum cholesterol	4.6 mmol/L	(< 5.2)

What is the most likely diagnosis?

(Please select 1 option)

○ Cerebellopontine angle tumour

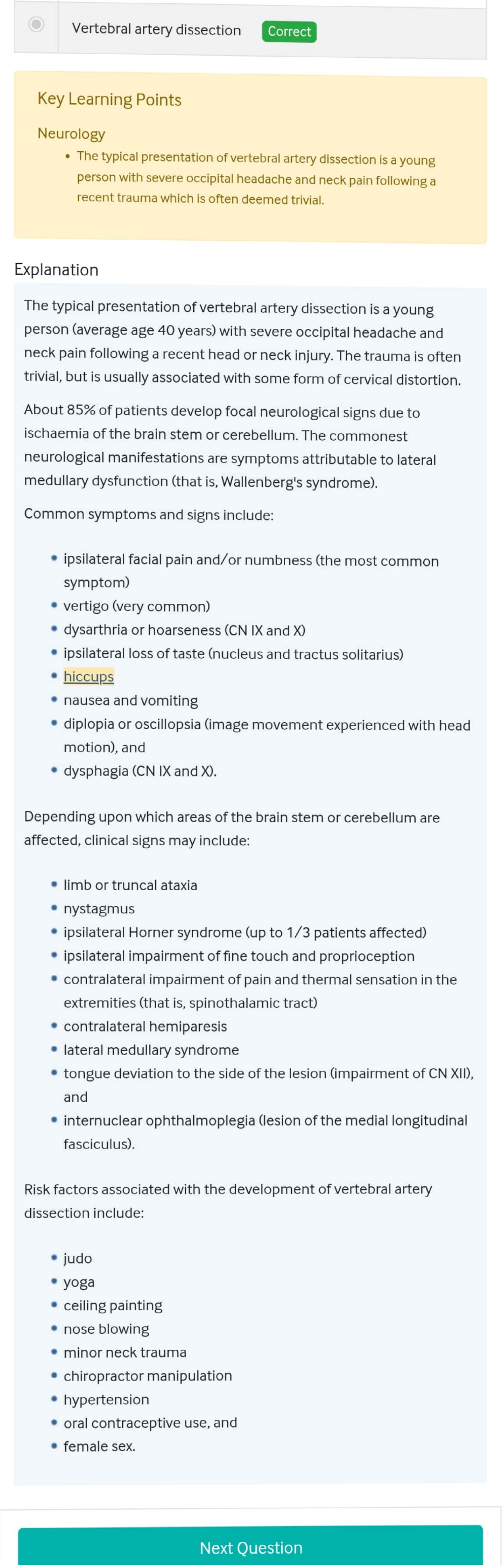
○ Cervical dislocation

○ Vertebrobasilar migraine

○ Subarachnoid haemorrhage

○ Vertebral artery dissection

Submit Answer



○ Vertebral artery dissection **Correct**

Key Learning Points

Neurology

- The typical presentation of vertebral artery dissection is a young person with severe occipital headache and neck pain following a recent trauma which is often deemed trivial.

Explanation

The typical presentation of vertebral artery dissection is a young person (average age 40 years) with severe occipital headache and neck pain following a recent head or neck injury. The trauma is often trivial, but is usually associated with some form of cervical distortion.

About 85% of patients develop focal neurological signs due to ischaemia of the brain stem or cerebellum. The commonest neurological manifestations are symptoms attributable to lateral medullary dysfunction (that is, Wallenberg's syndrome).

Common symptoms and signs include:

- ipsilateral facial pain and/or numbness (the most common symptom)
- vertigo (very common)
- dysarthria or hoarseness (CN IX and X)
- ipsilateral loss of taste (nucleus and tractus solitarius)
- hiccups
- nausea and vomiting
- diplopia or oscillopsia (image movement experienced with head motion), and
- dysphagia (CN IX and X).

Depending upon which areas of the brain stem or cerebellum are affected, clinical signs may include:

- limb or truncal ataxia
- nystagmus
- ipsilateral Horner syndrome (up to 1/3 patients affected)
- ipsilateral impairment of fine touch and proprioception
- contralateral impairment of pain and thermal sensation in the extremities (that is, spinothalamic tract)
- contralateral hemiparesis
- lateral medullary syndrome
- tongue deviation to the side of the lesion (impairment of CN XII), and
- internuclear ophthalmoplegia (lesion of the medial longitudinal fasciculus).

Risk factors associated with the development of vertebral artery dissection include:

- judo
- yoga
- ceiling painting
- nose blowing
- minor neck trauma
- chiropractor manipulation
- hypertension
- oral contraceptive use, and
- female sex.

Next Question

A 70-year-old man was admitted to the Emergency department having sustained a fall outside his home.

A neighbour who had witnessed the fall said that prior to the fall he had appeared unsteady on his feet and had staggered. He had banged his head on the pavement and had sustained a small laceration and occipital haematoma. The neighbour also commented that in the last week he had not seemed his normal self. He would normally come round on a Tuesday and they would go to the bingo hall together which had not happened. In addition he would also put the rubbish bags out for her and he had forgotten.

His past medical history included hypertension, hypercholesterolaemia, atrial fibrillation and mixed mitral valve disease. His regular medication included bendroflumethiazide, warfarin and simvastatin. He was a non-smoker and drank approximately 15 units of alcohol per week.

On examination he appeared dishevelled, aggressive and disorientated. His Glasgow coma scale was 13/15 (M6, E4, V3), blood pressure was 162/89 mmHg, pulse was 67 beats per minute and irregularly irregular and temperature was 37.1°C . Heart sounds revealed a harsh pan systolic murmur and displaced apex beat. Auscultation of the chest was normal. Neurological examination revealed an occipital haematoma. Cranial nerve examination revealed slurred speech. Peripheral nervous system examination revealed increased tone and reflexes on the right with a right extensor plantar. There was evidence of mild pyramidal weakness on the right and problems with coordination were generally observed.

Investigations revealed:

Sodium	136 mmol/L	(137-144)
Potassium	3.7 mmol/L	(3.5-4.9)
Urea	7.9 mmol/L	(2.5-7.5)
Creatinine	110 µmol/L	(60-110)
Haemoglobin	125 g/L	(130-180)
White cell count	10 ×10^9/L	(4-11)
Platelets	190 ×10^9/L	(150-400)
INR	4.2	(<1.4)

A CT scan of the brain (without contrast) showed mild generalised cerebral atrophy and a crescent-shaped heterogeneously dense lesion in the region of the left frontal lobe with a fluid level within the lesion. There was no evidence of occipital fracture.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Acute on chronic subdural haematoma
- ○ Subdural empyema
- ○ Normal pressure hydrocephalus
- ○ Epidural haematoma
- ○ Vertebrobasilar dissection

Submit Answer

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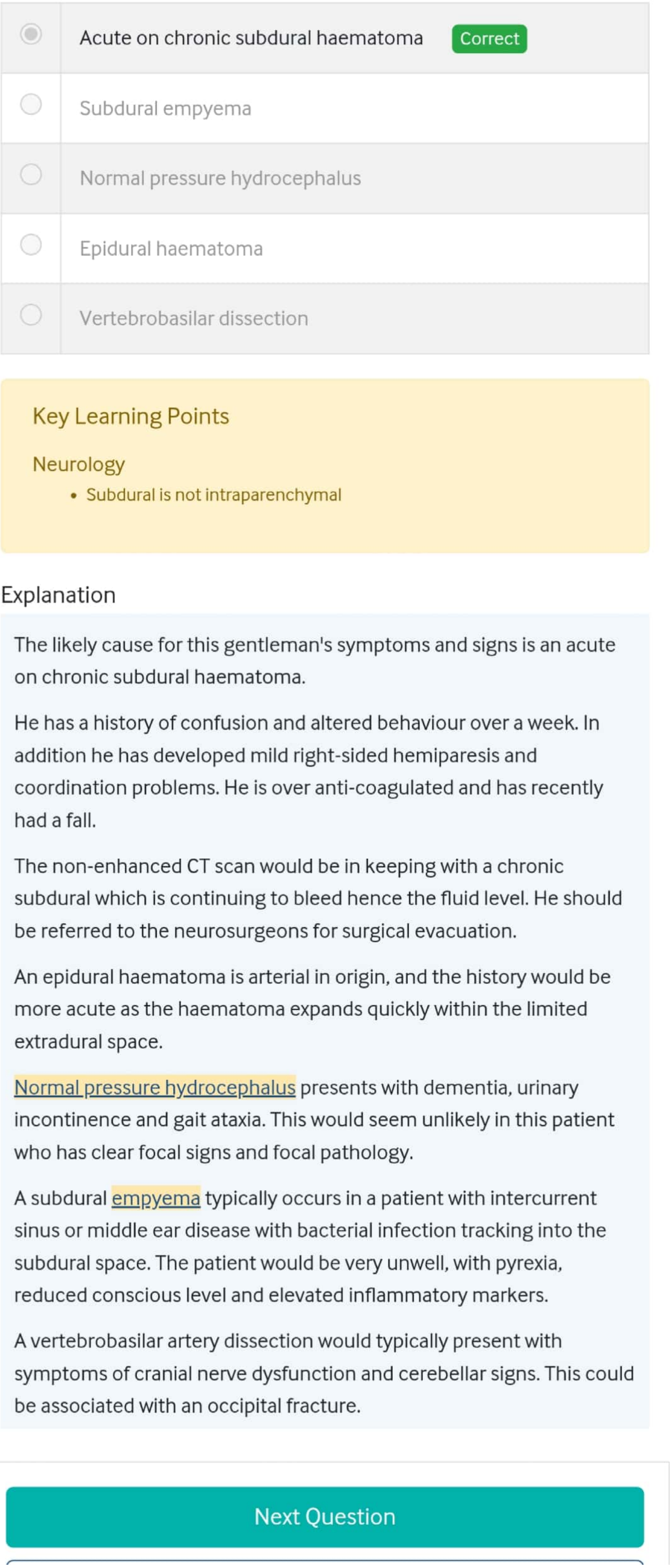

● Acute on chronic subdural haematoma

○ Subdural empyema

○ Normal pressure hydrocephalus

○ Epidural haematoma

○ Vertebrobasilar dissection

Key Learning Points

Neurology

- Subdural is not intraparenchymal

Explanation

The likely cause for this gentleman's symptoms and signs is an acute on chronic subdural haematoma.

He has a history of confusion and altered behaviour over a week. In addition he has developed mild right-sided hemiparesis and coordination problems. He is over anti-coagulated and has recently had a fall.

The non-enhanced CT scan would be in keeping with a chronic subdural which is continuing to bleed hence the fluid level. He should be referred to the neurosurgeons for surgical evacuation.

An epidural haematoma is arterial in origin, and the history would be more acute as the haematoma expands quickly within the limited extradural space.

Normal pressure hydrocephalus presents with dementia, urinary incontinence and gait ataxia. This would seem unlikely in this patient who has clear focal signs and focal pathology.

A subdural empyema typically occurs in a patient with intercurrent sinus or middle ear disease with bacterial infection tracking into the subdural space. The patient would be very unwell, with pyrexia, reduced conscious level and elevated inflammatory markers.

A vertebrobasilar artery dissection would typically present with symptoms of cranial nerve dysfunction and cerebellar signs. This could be associated with an occipital fracture.

You are referred an 18-year-old female with restricted eye movements and progressive muscle weakness.

At the age of 6 years she was noted to have a squint and was given glasses. At 7 she complained of double vision that gradually resolved. She did reasonably well until her early teens when she found herself unable to compete with other children at play. For the last one year her double vision has recurred and she has noticed progressive generalised weakness, for example difficulty rising from a seated to standing position.

On examination she was of short stature with ptosis and a paucity of spontaneous facial expressions. Mental status examination was normal. Eye movements were absent in all directions. The pupils responded to light. The visual fields were full to confrontation but her visual acuity was reduced even with correction. The fundi showed pigmentary degeneration. Cataracts were not present. Hearing was normal.

Motor examination revealed weakness of neck muscles and proximal muscle groups in the lower extremities. Deep tendon reflexes were reduced and plantar reflexes were flexor. Cerebellar testing revealed intact finger to nose, slow rapid alternating movements in the upper extremities with mild ataxia, moderate heel to shin ataxia and gait ataxia. Romberg's testing was steady with eyes open and closed. Sensory examination showed preserved sensation to all primary modalities. The rest of the systemic examination was unremarkable. ECG showed incomplete heart block.

Which of the following tests would be most helpful in establishing the diagnosis?

(Please select 1 option)

○ MRI brain

○ Urinary copper excretion

○ Muscle biopsy

○ Ambulatory EEG

○ Cerebrospinal fluid examination for oligoclonal bands

Submit Answer

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○ Urinary copper excretion

● Muscle biopsy **Correct**

○ Ambulatory EEG

○ Cerebrospinal fluid examination for oligoclonal bands

Key Learning Points

Neurology

- Even though rare, its important to be aware of the genetic mitochondrial disorders. Red ragged fibres are seen on muscle biopsy of Kearns Sayre syndrome.

Explanation

Kearns-Sayre syndrome produces the classic triad of:

- Progressive external ophthalmoplegia
- Pigmentary degeneration of the retina, and
- Heart block.

Most cases have large mitochondrial DNA mutations.

Ragged-red fibres are seen on muscle biopsy.

Although increased cerebrospinal fluid protein concentrations on lumbar puncture and white matter damage on MRI are seen, these changes are not specific to the syndrome.

Cortical blindness is not a complication of the condition.

Next Question

A 45-year-old woman presented to her GP complaining of tingling in the right palm and right wrist pain.

She had experienced these symptoms intermittently however recently they had been keeping her awake all night. She had noticed a reduction in grip and was struggling to work on the checkout counter at her local supermarket. Otherwise she felt well in herself and had not noticed any weakness in the other hand or lower limb.

Her weight was stable and she denied any neck problems or swallowing difficulties. She had a past medical history of hypothyroidism and hypertension and took regular thyroxine, bendroflumethiazide and ibuprofen. She was a non-smoker and rarely drank alcohol.

On examination she appeared alert and orientated. Fundoscopy and cranial nerve examination were all normal and neck movements were full.

On examination of the upper limb, there was wasting over the right thenar eminence and fasciculations with a small burn over the right index finger. No other fasciculations could be detected in the proximal limb or other hand. Tone appeared normal and reflexes were intact. There was weakness of thumb abduction and opposition, with loss of pinprick and light touch sensation over the thumb, index and middle finger in the right hand. On examination of the lower limb, no abnormalities could be found.

Nerve conduction studies showed absent sensory action potential in right median palmar branches and denervation of the right abductor pollicis brevis. Sensory and motor conduction studies in the left upper limb and lower limbs were normal.

What is the most likely diagnosis?

(Please select 1 option)

○ Ulnar nerve palsy

○ Thoracic inlet syndrome

○ Median nerve palsy

○ Motor neurone disease

○ Syringomyelia

Submit Answer

● Median nerve palsy Correct

○ Motor neurone disease

○ Syringomyelia

Key Learning Points

Neurology

- Carpal tunnel syndrome (CTS) is a result of pressure on the median nerve in the carpal tunnel.

Explanation

The history is in keeping with carpal tunnel syndrome (CTS) arising as a result of pressure on the median nerve in the carpal tunnel.

The median nerve supplies the muscles of the thenar eminence: the abductor pollicis (C7, C8), flexor pollicis brevis and opponens pollicis, and the lateral two lumbricals. The nerve conduction studies confirm marked denervation and absent sensory potentials within the median nerve territory.

CTS is associated with hypothyroidism, pregnancy, amyloidosis and rheumatoid arthritis.

There are no features to suggest motor neurone disease, which presents with a combination of both upper and lower motor neurone abnormalities in absence of sensory disturbance.

Syringomyelia within the cervical cord would cause lower motor neurone signs at the level of the syrinx, with dissociated pain and temperature loss and upper motor neurone signs in the legs.

Thoracic inlet syndrome affecting C8, T1 of the brachial plexus would cause additional weakness of hand muscles.

Ulnar nerve palsy would cause weakness of the small muscles of the hand with preserved thenar muscle function.

Next Question

A 56-year-old female presented to her GP with pain in the left side of her neck radiating down the lateral aspect of her arm and forearm.

She had also noticed some weakness of her left shoulder and struggled to elevate her arm. She had a long-standing history of rheumatoid arthritis, treated with steroids and penicillamine. She was a non-smoker and did not drink any alcohol.

On examination there was some wasting over the left deltoid and evidence of fasciculations. Neck movements appeared full except that lateral movement exacerbated the left arm pain.

On examination of the upper limb, tone appeared reduced at the elbow and wrist and the biceps jerk was only present on re-enforcement. The left supinator jerk was inverted and the triceps jerk appeared brisk. There was some weakness of left shoulder abduction, elbow flexion and supination, but finger movements and elbow extension were intact. There was a sensory deficit over the lateral aspect of the left upper arm and forearm. The right arm appeared normal.

On examination of he lower limb, tone was increased, but power appeared normal. All reflexes were brisk and both plantar responses were extensor.

Investigations showed:

Haemoglobin	114 g/L	(130-180)
White cell count	3.4 ×10^9/L	(4-11)
Platelets	245 ×10^9/L	(150-400)
ESR (Westergren)	45 mm/1st hour	(0-15)
Serum sodium	145 mmol/L	(137-144)
Serum potassium	3.2 mmol/L	(3.5-4.9)
Serum urea	6.7 mmol/L	(2.5-7.5)
Serum creatinine	135 umol/L	(60-110)
Serum creatine kinase	178 U/L	(24-170)
Fasting plasma glucose	8.7 mmol/L	(3-6)

What is the most likely diagnosis?

(Please select 1 option)

- ○ Inclusion body myositis
- ○ Circumflex neuropathy
- ○ Motor neurone disease
- ○ Cervical myelopathy
- ○ Steroid-induced myopathy

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○ Cervical myelopathy **This is the correct answer**

○ Steroid-induced myopathy

Key Learning Points

Neurology

- Cervical myopathy is a common complication of rheumatoid arthritis.

Explanation

This patient has a cervical myelopathy, most likely as a result of her rheumatoid arthritis.

The likely level is C5/C6 given that there is weakness of the deltoid, biceps and supinator and the supinator jerk is inverted.

She also has long tract signs in her legs with hypertonia and hyperreflexia.

A circumflex neuropathy would account for the weakness and fasciculations of the deltoid, but would not explain the upper motor neurone signs in the legs, or weakness of the biceps and supinator.

Inclusion body myositis, an inflammatory myopathy, would present with bilateral often asymmetrical weakness, which has a tendency to affect distal musculature. The tendon reflexes would be normal and the creatine kinase would be normal or mildly elevated.

Motor neurone disease presents with upper and lower motor signs in absence of sensory disturbance.

A steroid induced myopathy would cause proximal weakness and wasting with normal reflexes and sensation.

Next Question

A 35-year-old gentleman, who is an office worker, is admitted to hospital with fevers, rigors and right-sided flank pain with a urine dipstick positive for nitrites and leukocytes.

His pulse is 120, blood pressure is 140/90 mmHg and temperature is 38.2°C. He is on no regular medications.

Clinically, he is diagnosed with pyelonephritis and is commenced on intravenous antibiotics for a few days. There is no history of childhood urinary tract infections renal calculi or previous surgical procedures.

His admission laboratory results reveal the following:

Haemoglobin	140 g/L	(130-180)
White cell count	18 ×10^9/L	(4-11)
C reactive protein	130 mg/L	(<2)
Creatinine	90 µmol/L	(60-110)

An ultrasound scan of the renal tract shows multiple cysts in both kidneys, characteristic of polycystic kidney disease. He makes a good recovery and is due to be discharged soon.

Having read about this condition, the patient asks about whether imaging of the brain is necessary to look for cerebral aneurysms. The patient has no history of headache or neurological symptoms, and there is no family history of stroke or aneurysms.

Which is the most appropriate management step?

(Please select 1 option)

- ○ MRI brain as an outpatient
- ○ No routine imaging is required at this stage
- ○ CT brain as an inpatient
- ○ MRA of the brain as an outpatient
- ○ CT brain with intravenous contrast as an outpatient

Submit Answer

● No routine imaging is required at this stage **Correct**

○ CT brain as an inpatient

○ MRA of the brain as an outpatient

○ CT brain with intravenous contrast as an outpatient

Key Learning Points

Nephrology, Neurology

- Patients with adult polycystic kidney disease should only have screening for cerebral aneurysms if high risk (ie previous ruptured aneurysm, neurological symptoms or positive FHx).

Explanation

It is estimated that up to 8% of patients with adult polycystic kidney disease have cerebral aneurysms.

Current consensus is that screening for cerebral aneurysms should only be carried out in high risk patients. These include factors such as:

- previous rupture of aneurysm
- concerning neurological symptoms (for example, severe headache)
- positive family history of haemorrhagic stroke or aneurysm.

Even if aneurysms are found, the rupture risk can still be low and the morbidity implications of curative surgery may outweigh conservative management.

This patient does not have any risk factors and so no routine screening is required.

A 50-year-old man presents with tingling in the left upper limb. The pain originated in the neck and radiated down the left arm. He proceeded to have numbness and paraesthesia in the left lower limb.

On examination he had restriction of neck movements and there was a mild wasting to be noted in the left biceps. There was inversion of the supinator and biceps jerks. His knee jerk and ankle jerk were hyperreactive and he has a positive extensor plantar response.

He then developed paraesthesia and numbness of the right lower limb. A diagnosis of cord compression was made and he underwent a surgical decompression.

Post-surgery was complicated by septicaemia and urinary tract infection and he remained in bed for four days. He subsequently developed inability to dorsiflex his right foot and right big toe. There was numbness on the outside of the foot and there was decreased eversion, but inversion was normal. His reflexes remained as before.

What is the cause of the post operative weakness?

(Please select 1 option)

- ○ Common peroneal nerve palsy
- ○ L4 root lesion
- ○ Sciatic nerve palsy
- ○ Spinal cord infarction
- ○ Recurrence of the original cord compression

Submit Answer

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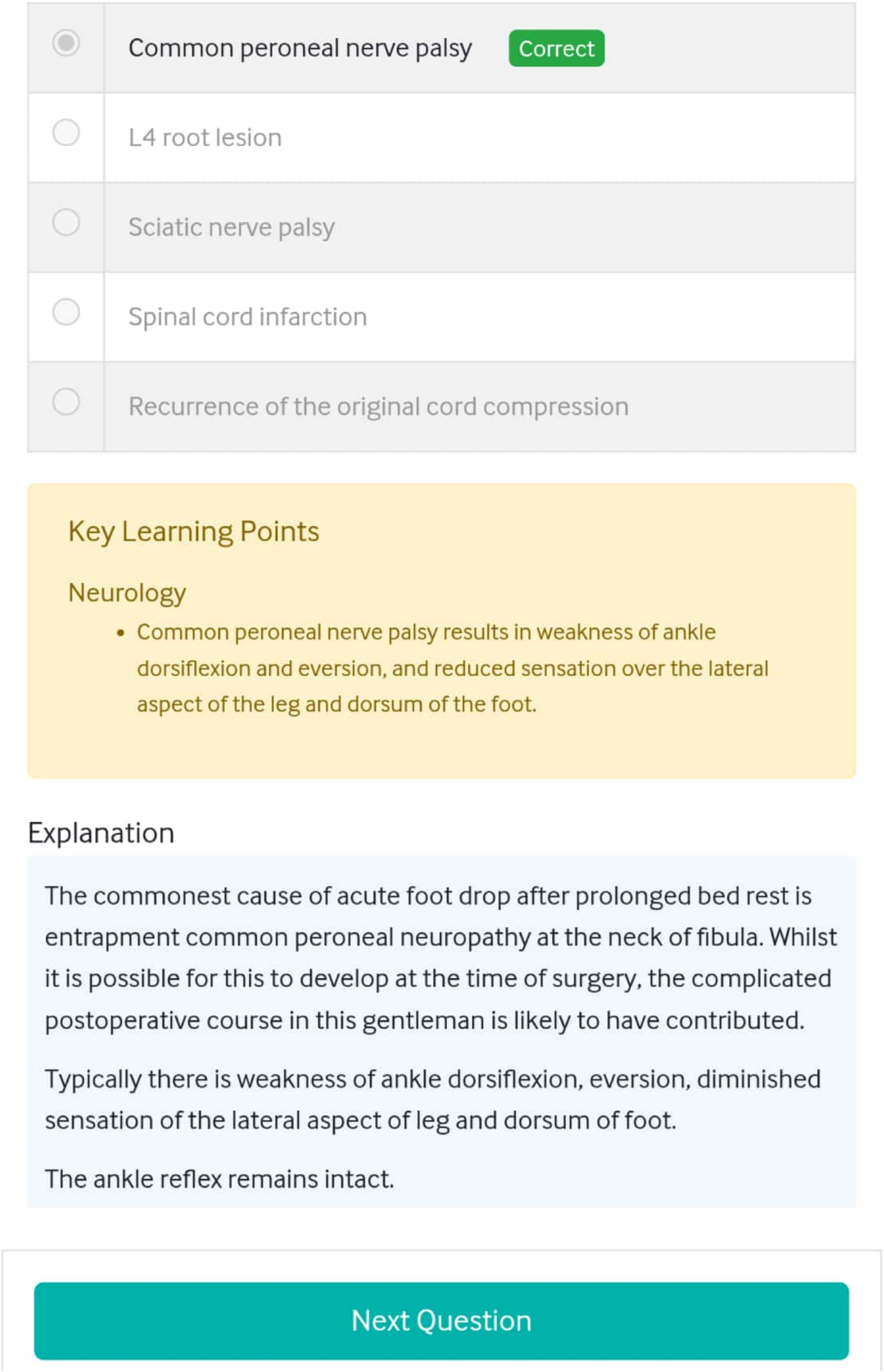


● Common peroneal nerve palsy **Correct**

○ L4 root lesion

○ Sciatic nerve palsy

○ Spinal cord infarction

○ Recurrence of the original cord compression

Key Learning Points

Neurology

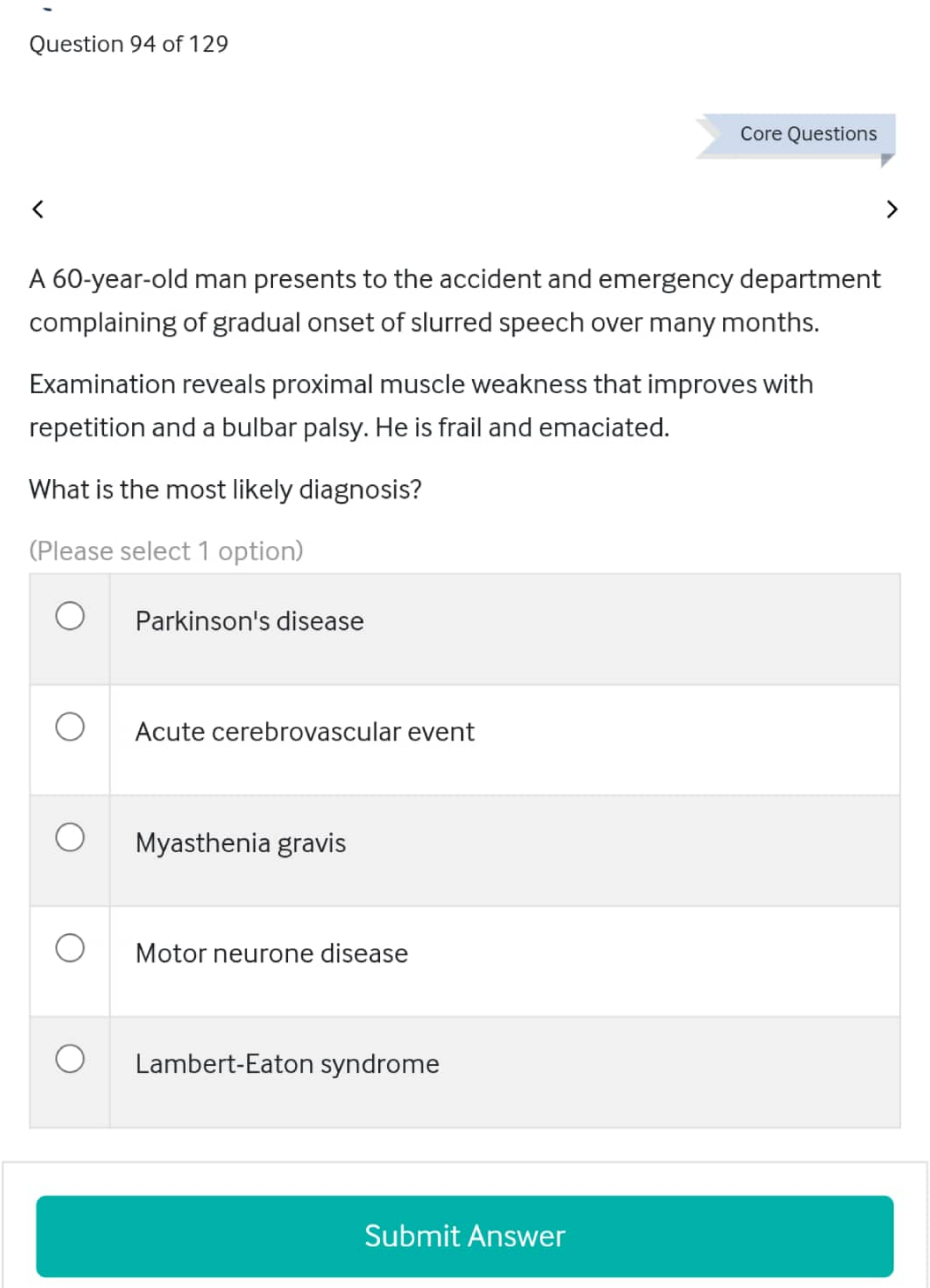
- Common peroneal nerve palsy results in weakness of ankle dorsiflexion and eversion, and reduced sensation over the lateral aspect of the leg and dorsum of the foot.

Explanation

The commonest cause of acute foot drop after prolonged bed rest is entrapment common peroneal neuropathy at the neck of fibula. Whilst it is possible for this to develop at the time of surgery, the complicated postoperative course in this gentleman is likely to have contributed.

Typically there is weakness of ankle dorsiflexion, eversion, diminished sensation of the lateral aspect of leg and dorsum of foot.

The ankle reflex remains intact.



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A 60-year-old man presents to the accident and emergency department complaining of gradual onset of slurred speech over many months.

Examination reveals proximal muscle weakness that improves with repetition and a bulbar palsy. He is frail and emaciated.

What is the most likely diagnosis?

(Please select 1 option)

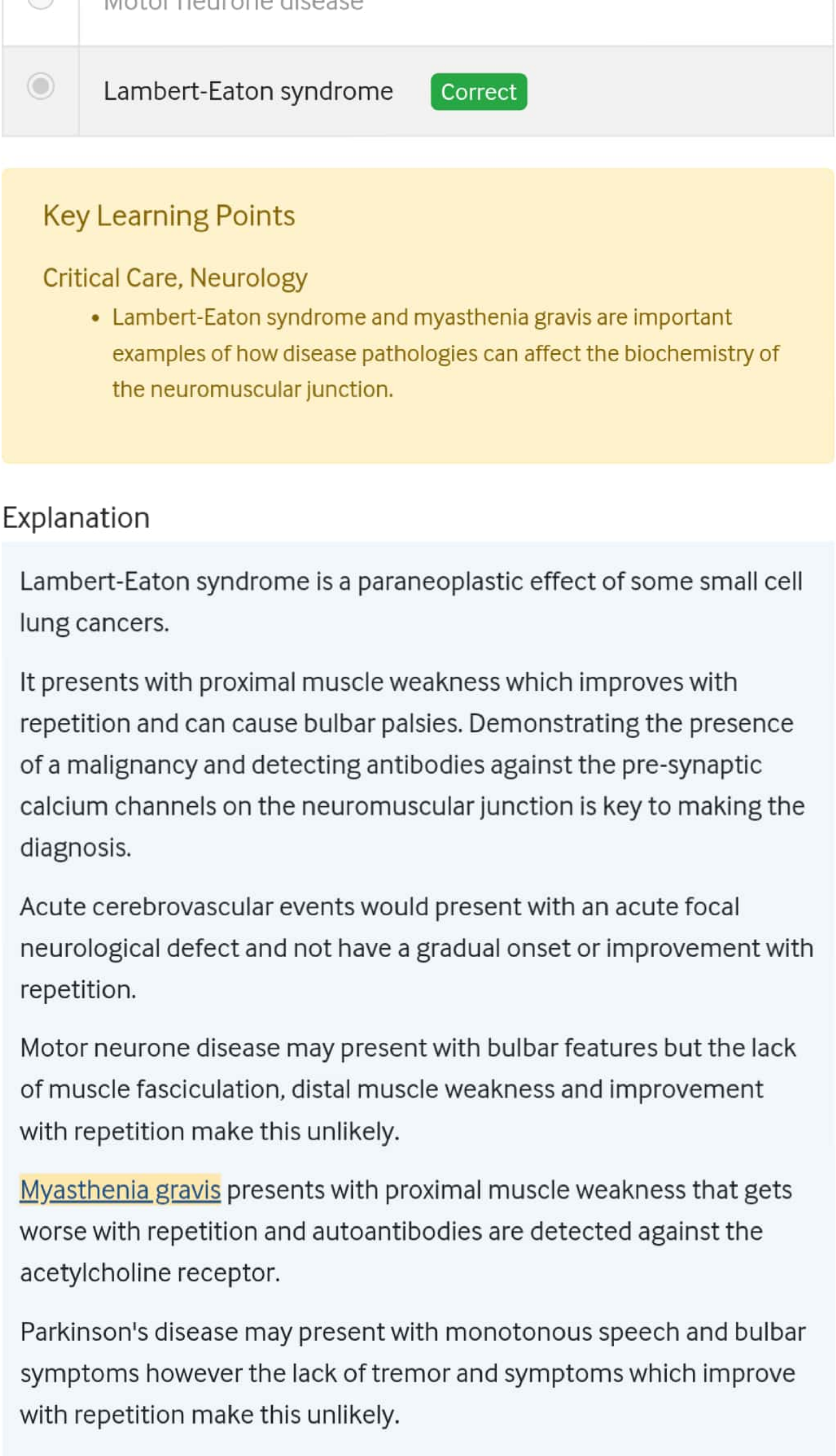
○ Parkinson's disease

○ Acute cerebrovascular event

○ Myasthenia gravis

○ Motor neurone disease

○ Lambert-Eaton syndrome



○ Motor neurone disease

● Lambert-Eaton syndrome **Correct**

Key Learning Points

Critical Care, Neurology

- Lambert-Eaton syndrome and myasthenia gravis are important examples of how disease pathologies can affect the biochemistry of the neuromuscular junction.

Explanation

Lambert-Eaton syndrome is a paraneoplastic effect of some small cell lung cancers.

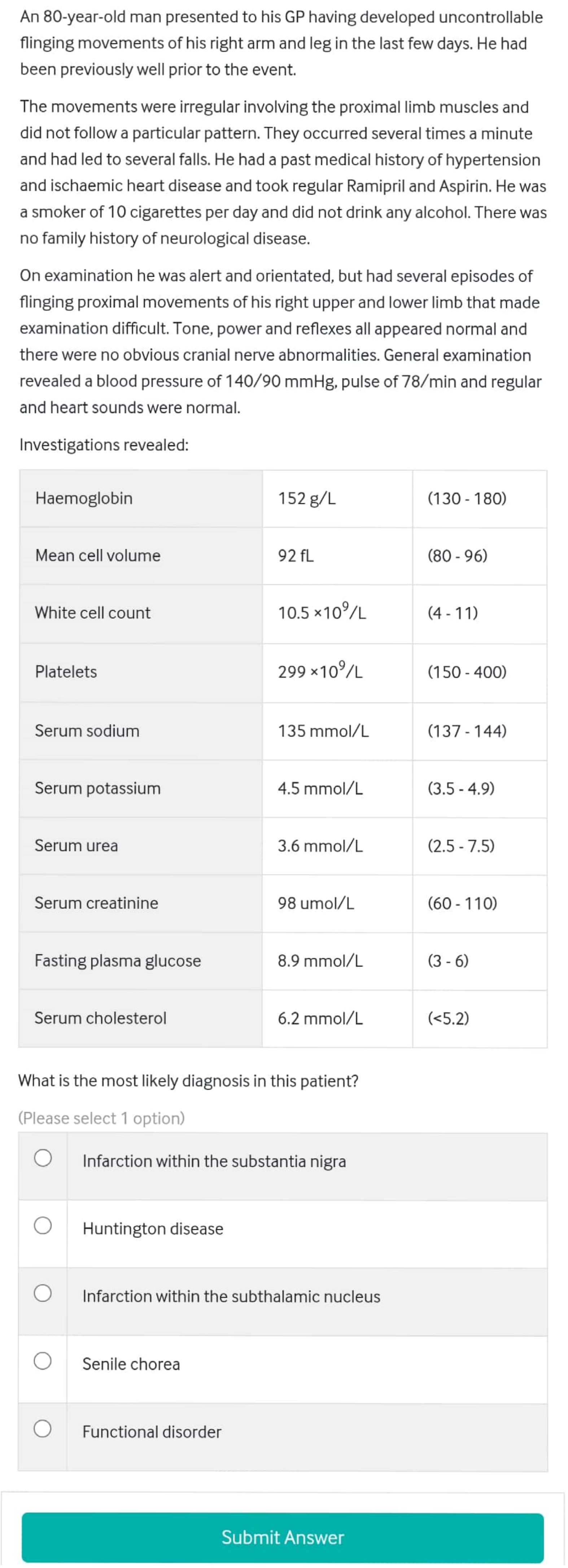
It presents with proximal muscle weakness which improves with repetition and can cause bulbar palsies. Demonstrating the presence of a malignancy and detecting antibodies against the pre-synaptic calcium channels on the neuromuscular junction is key to making the diagnosis.

Acute cerebrovascular events would present with an acute focal neurological defect and not have a gradual onset or improvement with repetition.

Motor neurone disease may present with bulbar features but the lack of muscle fasciculation, distal muscle weakness and improvement with repetition make this unlikely.

Myasthenia gravis presents with proximal muscle weakness that gets worse with repetition and autoantibodies are detected against the acetylcholine receptor.

Parkinson's disease may present with monotonous speech and bulbar symptoms however the lack of tremor and symptoms which improve with repetition make this unlikely.



An 80-year-old man presented to his GP having developed uncontrollable flinging movements of his right arm and leg in the last few days. He had been previously well prior to the event.

The movements were irregular involving the proximal limb muscles and did not follow a particular pattern. They occurred several times a minute and had led to several falls. He had a past medical history of hypertension and ischaemic heart disease and took regular Ramipril and Aspirin. He was a smoker of 10 cigarettes per day and did not drink any alcohol. There was no family history of neurological disease.

On examination he was alert and orientated, but had several episodes of flinging proximal movements of his right upper and lower limb that made examination difficult. Tone, power and reflexes all appeared normal and there were no obvious cranial nerve abnormalities. General examination revealed a blood pressure of 140/90 mmHg, pulse of 78/min and regular and heart sounds were normal.

Investigations revealed:

Haemoglobin	152 g/L	(130 - 180)
Mean cell volume	92 fL	(80 - 96)
White cell count	10.5 $\times 10^9$/L	(4 - 11)
Platelets	299 $\times 10^9$/L	(150 - 400)
Serum sodium	135 mmol/L	(137 - 144)
Serum potassium	4.5 mmol/L	(3.5 - 4.9)
Serum urea	3.6 mmol/L	(2.5 - 7.5)
Serum creatinine	98 umol/L	(60 - 110)
Fasting plasma glucose	8.9 mmol/L	(3 - 6)
Serum cholesterol	6.2 mmol/L	(<5.2)

What is the most likely diagnosis in this patient?

(Please select 1 option)

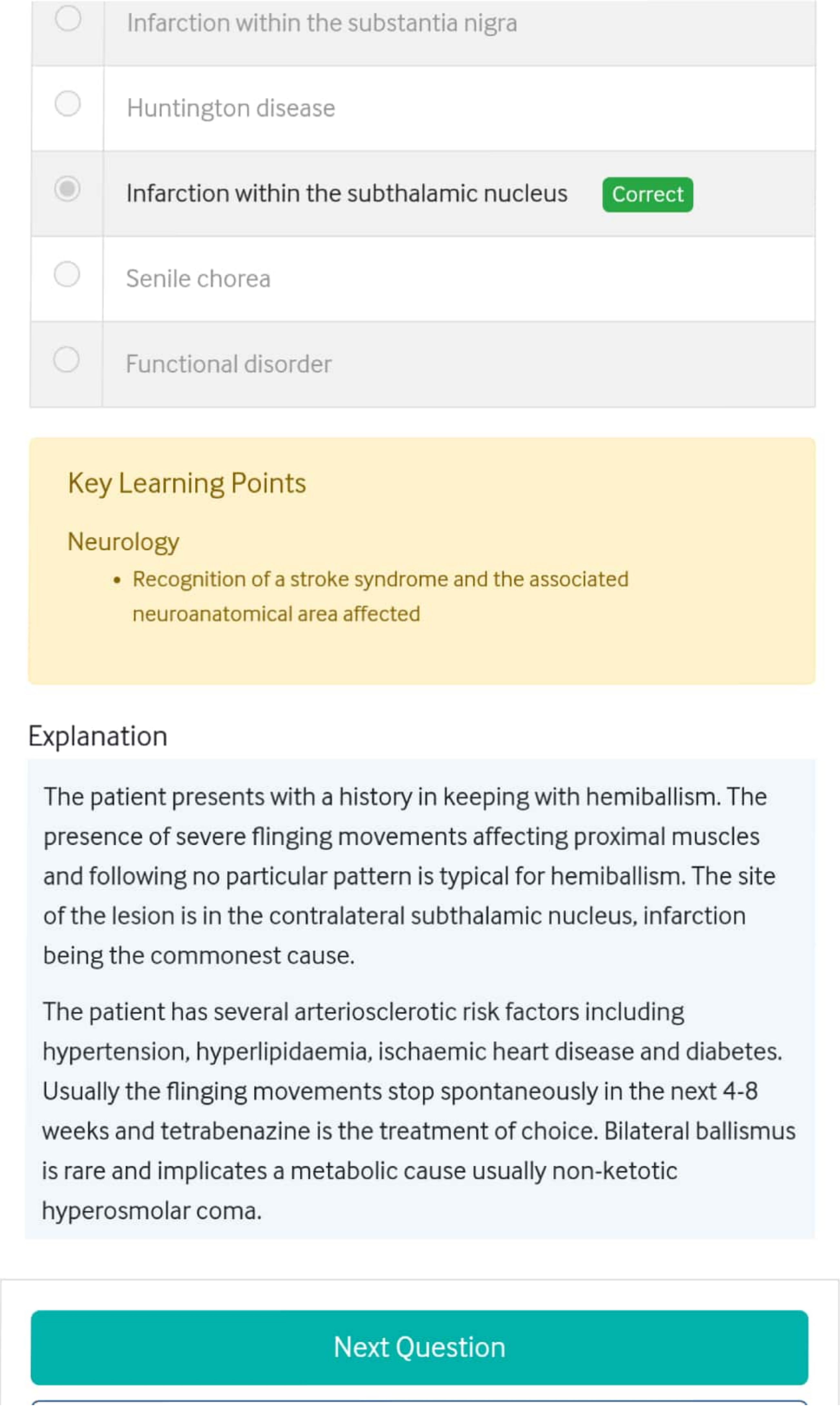
○ Infarction within the substantia nigra

○ Huntington disease

○ Infarction within the subthalamic nucleus

○ Senile chorea

○ Functional disorder



○ Infarction within the substantia nigra

○ Huntington disease

● Infarction within the subthalamic nucleus

○ Senile chorea

○ Functional disorder

Key Learning Points

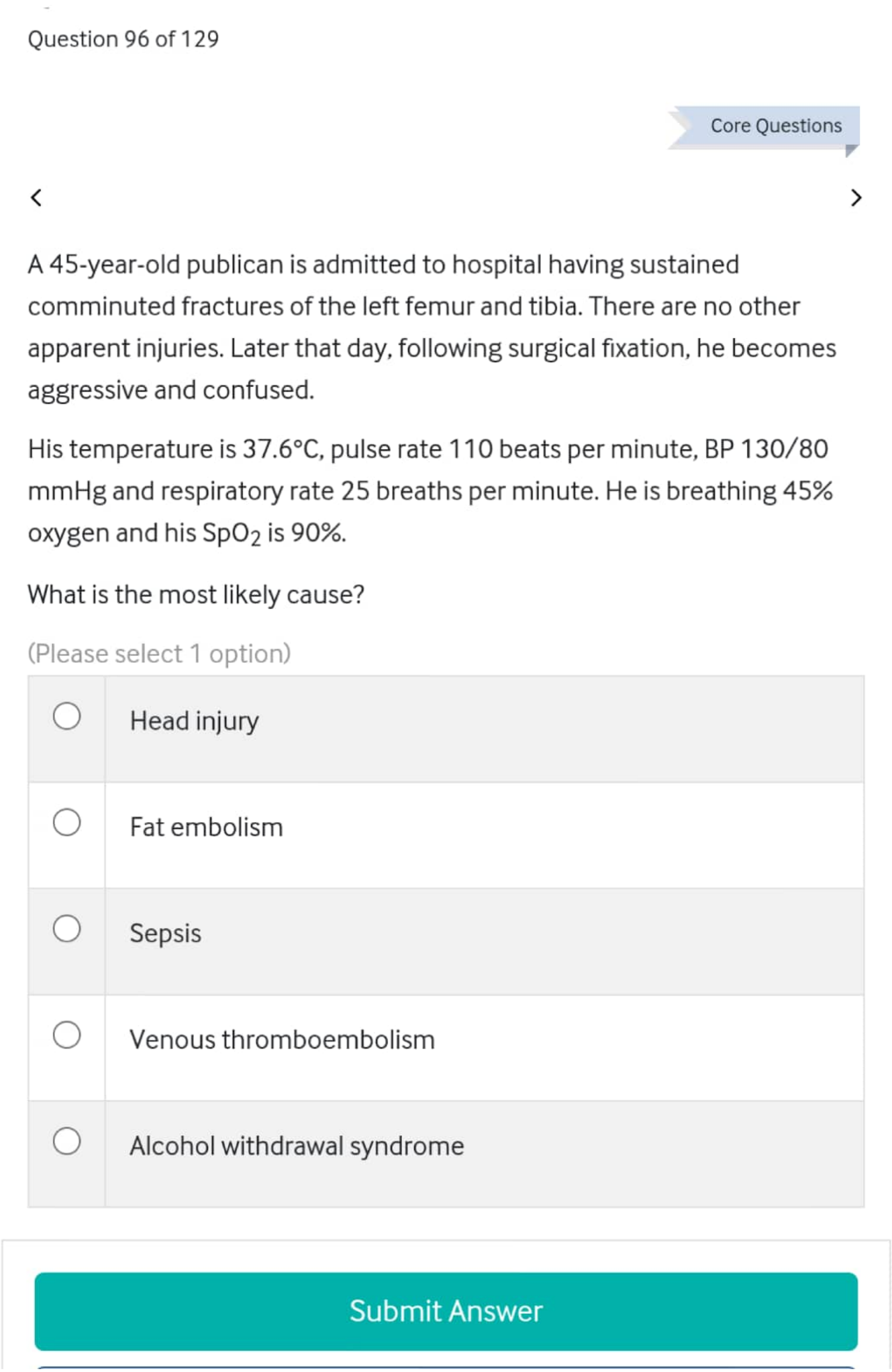
Neurology

- Recognition of a stroke syndrome and the associated neuroanatomical area affected

Explanation

The patient presents with a history in keeping with hemiballism. The presence of severe flinging movements affecting proximal muscles and following no particular pattern is typical for hemiballism. The site of the lesion is in the contralateral subthalamic nucleus, infarction being the commonest cause.

The patient has several arteriosclerotic risk factors including hypertension, hyperlipidaemia, ischaemic heart disease and diabetes. Usually the flinging movements stop spontaneously in the next 4-8 weeks and tetrabenazine is the treatment of choice. Bilateral ballismus is rare and implicates a metabolic cause usually non-ketotic hyperosmolar coma.



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A 45-year-old publican is admitted to hospital having sustained comminuted fractures of the left femur and tibia. There are no other apparent injuries. Later that day, following surgical fixation, he becomes aggressive and confused.

His temperature is 37.6°C, pulse rate 110 beats per minute, BP 130/80 mmHg and respiratory rate 25 breaths per minute. He is breathing 45% oxygen and his SpO_2 is 90%.

What is the most likely cause?

(Please select 1 option)

○ Head injury

○ Fat embolism

○ Sepsis

○ Venous thromboembolism

○ Alcohol withdrawal syndrome

○ Head injury

● Fat embolism Correct

○ Sepsis

○ Venous thromboembolism

○ Alcohol withdrawal syndrome

Key Learning Points

Critical Care, Neurology

- Fat embolism syndrome is a clinical diagnosis secondary to the presence of fat globules in the lung parenchyma and circulation that are released, typically following long bone fractures.

Explanation

The history and clinical features of this case suggest fat embolism syndrome. Fat embolism syndrome is a *clinical* diagnosis secondary to the presence of fat globules in the lung parenchyma and circulation that are typically released following long bone fractures. It requires high index of suspicion and usually presents 12-72 hours after initial injury.

There is a classic triad of:

- Respiratory changes - Dyspnoea, tachypnoea and hypoxaemia are early findings. These may progress to respiratory failure and ARDS requiring mechanical ventilation.
- Neurological features - Cerebral emboli produce neurological signs in up to 86% cases. They often occur after onset of respiratory symptoms, with a wide spectrum ranging from mild confusion and drowsiness to seizures. Usually there is an acute confusional state with or without focal signs. Most neurological deficits are transient and reversible.
- Petechial rash - This is the final component of the triad to develop and occurs in 60% of cases. Rash is seen in the conjunctiva, mucous membranes, skin folds of upper body particularly neck and axilla, appearing within the first 36 hours.

Patients may also develop:

- pyrexia
- tachycardia
- ECG changes (ST segment depression and right heart strain)
- fluffy retinal exudates
- coagulopathy, and
- renal changes (oliguria, lipiduria, proteinuria or haematuria).

The neurological symptoms might fit with alcohol withdrawal but the patient is unlikely to have the pyrexia and respiratory symptoms. Likewise, the development of sepsis, venous thromboembolism and the deterioration of a head injury do not fit with the clinical scenario.

Next Question

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A 28-year-old man was admitted to hospital following a car accident.

He had sustained significant back and chest injuries. Imaging of the spine revealed a cord compression at T3, and he was subsequently transferred to the neurosurgical unit for decompression. He was making a good recovery on the neuro-rehabilitation unit.

The on-call doctor was asked to see the patient as he had suddenly gone into urinary retention and required catheterisation. When the doctor arrived the patient was complaining of blurred vision, headaches and appeared to be in some distress.

Prior to his accident he had attended the GP for episodes of palpitations and weight loss. He did not take any regular prescribed medication but took ProPlus tablets when he had work deadlines. He worked in business and finance.

On examination he appeared to be profusely sweating and his face and shoulders were flushed. His blood pressure was 160/90 mmHg, pulse was 58/min and regular and temperature was 36.5°C. Heart and chest examinations were normal. Examination of the abdomen revealed fullness within the suprapubic area. Fundoscopy appeared normal, but during visual field testing the patient reported spots in both visual fields. The rest of the cranial nerves were intact. Examination of the peripheral nervous system revealed normal tone, power, reflexes, sensation and coordination in the upper limb, but increased tone, reflexes and pyramidal weakness together with diminished pinprick to the level of the nipple line.

Investigations revealed:

Haemoglobin	134 g/L	(130-180)
White cell count	10.9 $\times 10^9$/L	(4-11)
Platelets	180 $\times 10^9$/L	(150-400)
C reactive protein	10 mg/L	(<10)
Sodium	136 mmol/L	(137-144)
Potassium	3.8 mmol/L	(3.5-4.9)
Urea	7.9 mmol/L	(2.5-7.5)
Creatinine	88 μmol/L	(60-110)
Urinary vanillyl mandelic acid	35 mol/24h	(5-35)

What is the most likely diagnosis?

(Please select 1 option)

- ○ Autonomic dysreflexia (AD)
- ○ Anxiety attack
- ○ Essential hypertension
- ○ Pheochromocytoma
- ○ Spinal abscess with meningitis

Submit Answer

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(Please select 1 option)

- ○ Autonomic dysreflexia (AD) **This is the correct answer**
- ● Anxiety attack **Incorrect answer selected**
- ○ Essential hypertension
- ○ Pheochromocytoma
- ○ Spinal abscess with meningitis

Key Learning Points

Neurology

- Autonomic dysreflexia (AD) is a syndrome of massive imbalanced reflex sympathetic discharge occurring in patients with spinal cord injury (SCI)

Explanation

This patient has presented with symptoms of sympathetic overactivity (sweating, anxiety, headaches, blurred vision and elevated systolic blood pressure) in the presence of a known spinal cord injury above T6.

The diagnosis is autonomic dysreflexia (AD). Autonomic dysreflexia (AD) is a syndrome of massive imbalanced reflex sympathetic discharge occurring in patients with spinal cord injury (SCI) above the splanchnic sympathetic outflow (T5-T6).

Below the injury, intact peripheral sensory nerves transmit impulses that ascend in the spinothalamic and posterior columns to stimulate sympathetic neurons located in the intermediolateral grey matter of the spinal cord. The inhibitory outflow above the SCI from cerebral vasomotor centres is increased, but it is unable to pass below the block of the SCI.

This large sympathetic outflow causes release of various neurotransmitters (not epinephrine and dopamine), causing piloerection, skin pallor, and severe vasoconstriction in arterial vasculature. The result is sudden elevation in blood pressure and vasodilation above the level of injury.

An antihypertensive agent with rapid onset and short duration (nifedipine) is administered for elevated blood pressure.

Next Question

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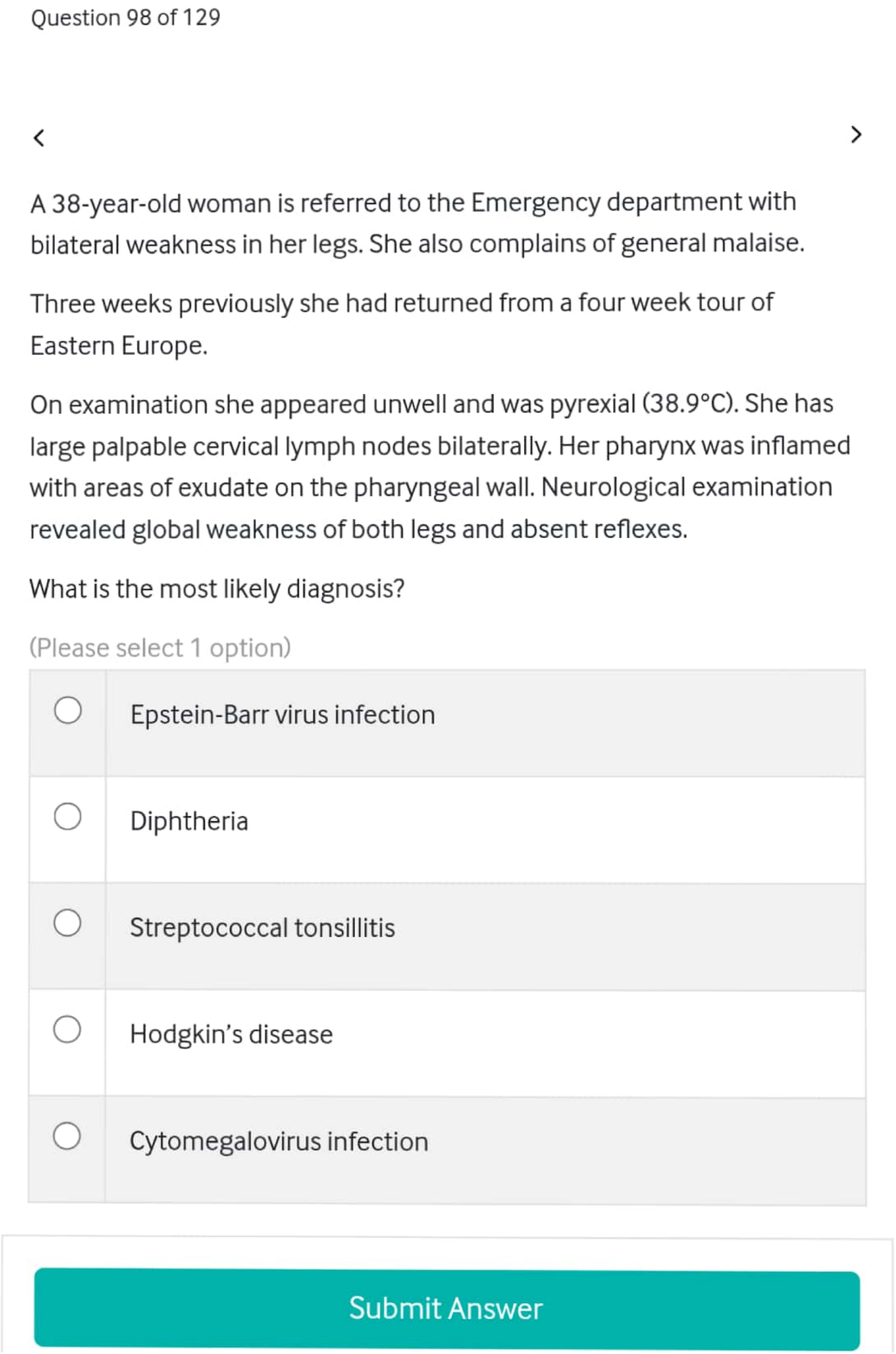

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A 38-year-old woman is referred to the Emergency department with bilateral weakness in her legs. She also complains of general malaise.

Three weeks previously she had returned from a four week tour of Eastern Europe.

On examination she appeared unwell and was pyrexial (38.9°C). She has large palpable cervical lymph nodes bilaterally. Her pharynx was inflamed with areas of exudate on the pharyngeal wall. Neurological examination revealed global weakness of both legs and absent reflexes.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Epstein-Barr virus infection
- ○ Diphtheria
- ○ Streptococcal tonsillitis
- ○ Hodgkin’s disease
- ○ Cytomegalovirus infection

○ Epstein-Barr virus infection

● Diphtheria **Correct**

○ Streptococcal tonsillitis

○ Hodgkin's disease

○ Cytomegalovirus infection

Key Learning Points

Infectious Diseases, Neurology

- Pharyngeal diphtheria presents with fever, sore throat, cervical lymphadenopathy and an adherent, greyish pharyngeal membrane.

Explanation

The breakdown of healthcare services in the former USSR was associated with a major resurgence of diphtheria.

Pharyngeal diphtheria presents with:

- fever
- sore throat
- cervical lymphadenopathy, and
- an adherent, grayish pharyngeal membrane.

The diphtheria toxin causes cardio- and neurotoxicity.

Treatment consists of antibiotic therapy and diphtheria antitoxin.

Next Question

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A 66-year-old woman was seen by her GP with a history of sudden onset right visual disturbance associated with right temporal throbbing headache.

She described the visual disturbance as a moving silhouette descending from above leading to complete visual loss. This lasted approximately one minute and resolved.

In addition she complained of pain when combing her hair and also pain in her jaw when chewing food. This had happened several times in the last week. She had a past history of polymyalgia rheumatica and hypertension. Her regular medication was bendroflumethiazide 2.5 mg/day.

On examination she was alert and orientated. Her blood pressure was 140/75 mmHg and pulse was 76/min and regular. She was apyrexial. On cardiovascular examination she had an ejection systolic murmur radiating into the neck and no carotid bruits. Fundoscopy examination revealed bilateral silver wiring and arteriovenous nipping. Pupils were equal and reactive and the rest of the cranial nerves were normal. The right temporal/jaw area appeared tender and pulsatile. Peripheral nervous system examination was normal.

Blood tests:

Haemoglobin	106 g/L	(115-165)
White cell count	5.6 $\times 10^9$/L	(4-11)
Platelets	500 $\times 10^9$/L	(150-400)
Mean cell volume	86 fL	(80-96)
Erythrocyte sedimentation rate	100 mm/1st hr	(0-30)
Cholesterol	6.2 mmol/L	(<5.2)
Sodium	134 mmol/L	(137-144)
Potassium	4.0 mmol/L	(3.5-4.9)
Urea	5.6 mmol/L	(2.5-7.5)
Creatinine	120 μmol/L	(60-110)
Glucose	5.7 mmol/L	(3.0-6.0)

Carotid Dopplers:

Right internal carotid artery	<50% stenosis
Left internal carotid artery	<50% stenosis

Given the above history and clinical findings, how should this patient be managed?

(Please select 1 option)

- ○ Commence on carbamazepine 300 mg/day
- ○ Commence on prednisolone 60 mg/day
- ○ Arrange for right internal carotid endarterectomy
- ○ Commence on aspirin 75 mg/day
- ○ Treat with sumatriptan

Submit Answer

○ Commence on prednisolone 60 mg/day
This is the correct answer

● Arrange for right internal carotid endarterectomy
Incorrect answer selected

○ Commence on aspirin 75 mg/day

○ Treat with sumatriptan

Key Learning Points

Neurology

- Treatment of TA includes high dose steroids followed by biopsy to confirm diagnosis

Explanation

This patient presents with a classic history of amaurosis fugax secondary to giant cell arteritis (GCA).

She complains of descending visual loss, jaw claudication associated with temporal headache and tenderness.

Her erythrocyte sedimentation rate (ESR) and platelets are elevated and she has a normocytic anaemia. She also has evidence of arteriosclerosis and probable aortic stenosis. She is therefore at high risk of thromboembolic events as well.

Her blood pressure is under control at present however she has elevated cholesterol, which should be addressed.

Treatment with prednisolone should be commenced immediately as there is a large threat to vision in the right eye. One should attempt to obtain tissue in order to achieve a diagnosis of GCA and initially should be from the affected side. Repeat ESR levels are used to monitor the condition.

An ECHO is indicated, but not in the first instance. There is no clinical or paraclinical evidence to suggest a septic thromboembolic event secondary to subacute bacterial endocarditis.

Next Question

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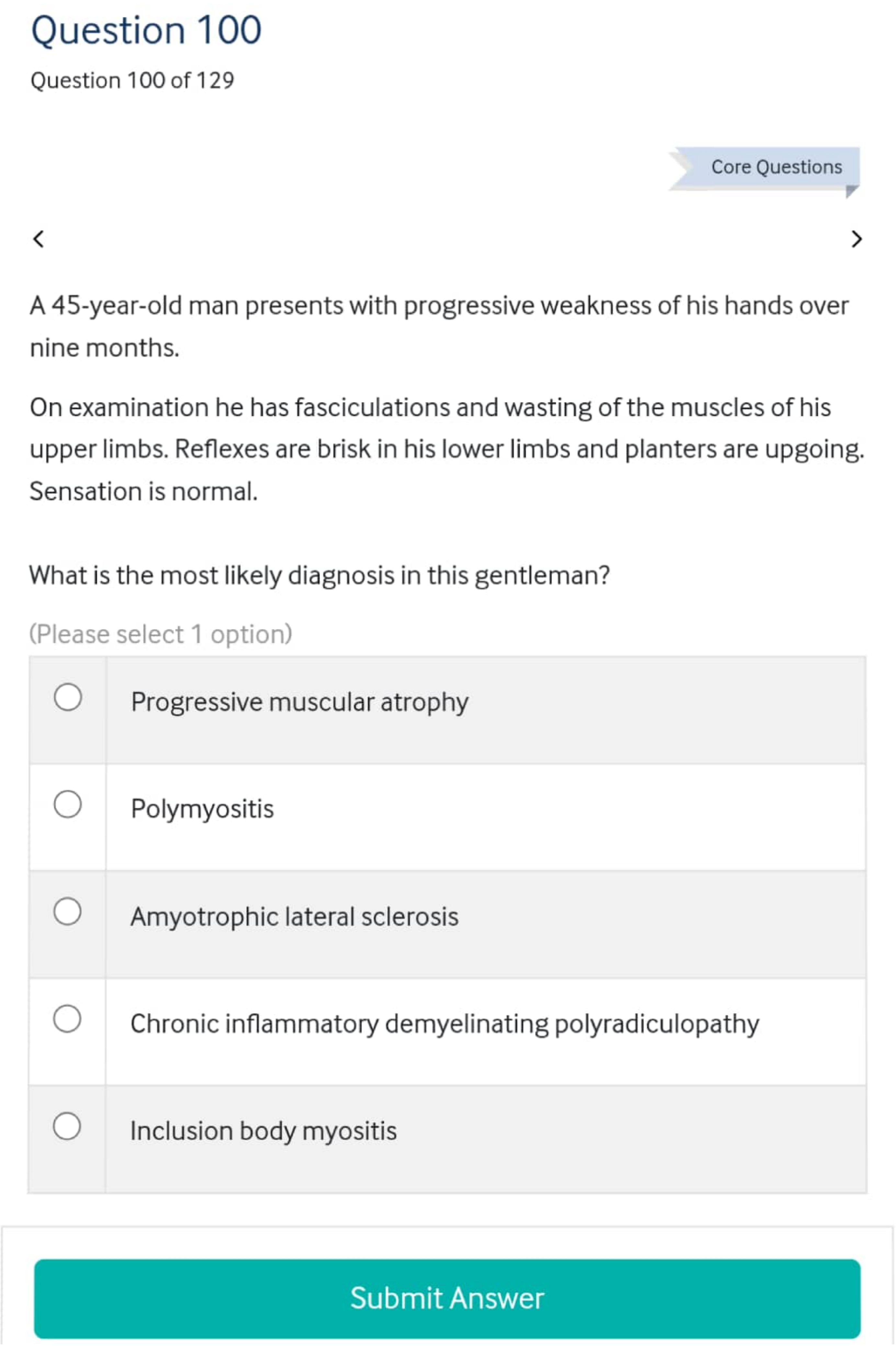

Question 100

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Core Questions

A 45-year-old man presents with progressive weakness of his hands over nine months.

On examination he has fasciculations and wasting of the muscles of his upper limbs. Reflexes are brisk in his lower limbs and planters are upgoing. Sensation is normal.

What is the most likely diagnosis in this gentleman?

(Please select 1 option)

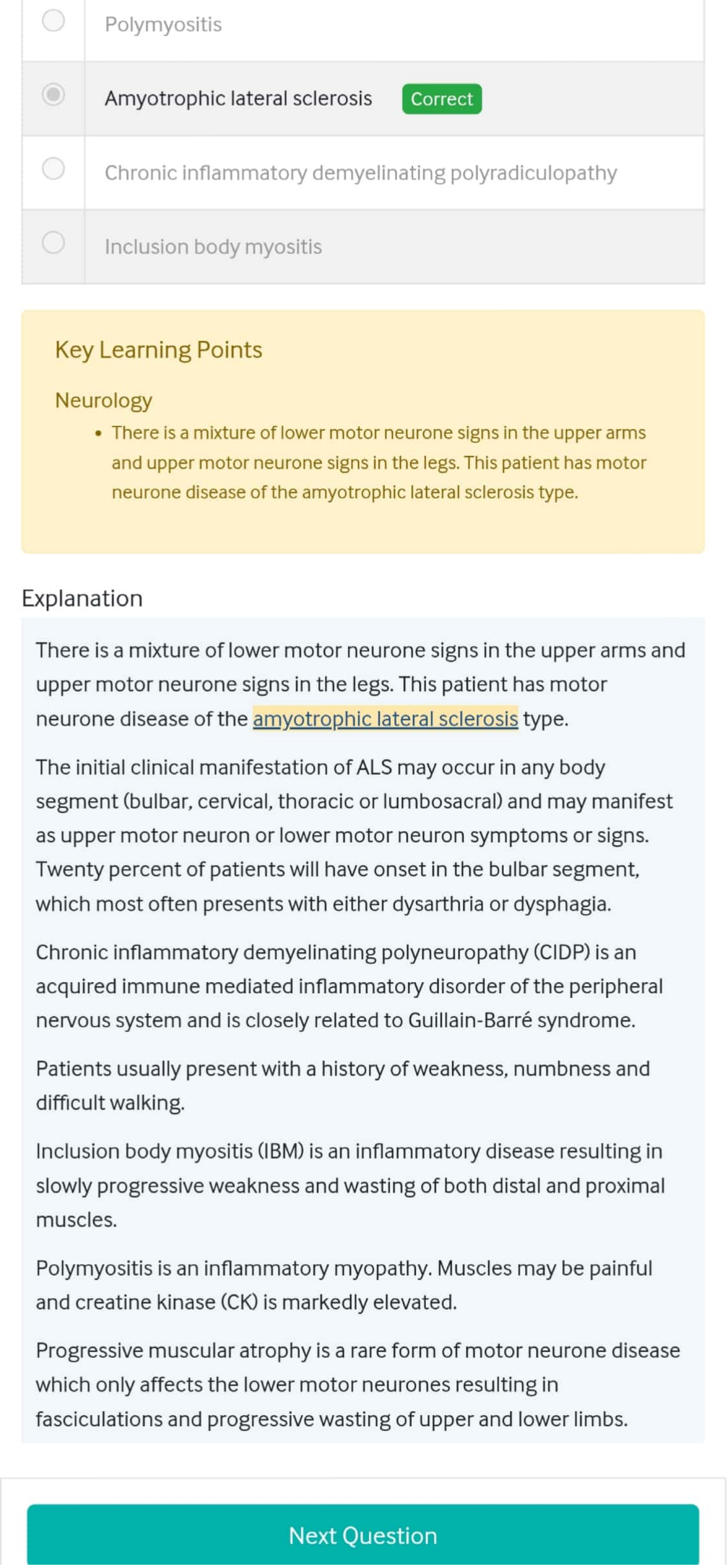
○ Progressive muscular atrophy

○ Polymyositis

○ Amyotrophic lateral sclerosis

○ Chronic inflammatory demyelinating polyradiculopathy

○ Inclusion body myositis



○ Polymyositis

● Amyotrophic lateral sclerosis **Correct**

○ Chronic inflammatory demyelinating polyradiculopathy

○ Inclusion body myositis

Key Learning Points

Neurology

- There is a mixture of lower motor neurone signs in the upper arms and upper motor neurone signs in the legs. This patient has motor neurone disease of the amyotrophic lateral sclerosis type.

Explanation

There is a mixture of lower motor neurone signs in the upper arms and upper motor neurone signs in the legs. This patient has motor neurone disease of the amyotrophic lateral sclerosis type.

The initial clinical manifestation of ALS may occur in any body segment (bulbar, cervical, thoracic or lumbosacral) and may manifest as upper motor neuron or lower motor neuron symptoms or signs. Twenty percent of patients will have onset in the bulbar segment, which most often presents with either dysarthria or dysphagia.

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an acquired immune mediated inflammatory disorder of the peripheral nervous system and is closely related to Guillain-Barré syndrome.

Patients usually present with a history of weakness, numbness and difficult walking.

Inclusion body myositis (IBM) is an inflammatory disease resulting in slowly progressive weakness and wasting of both distal and proximal muscles.

Polymyositis is an inflammatory myopathy. Muscles may be painful and creatine kinase (CK) is markedly elevated.

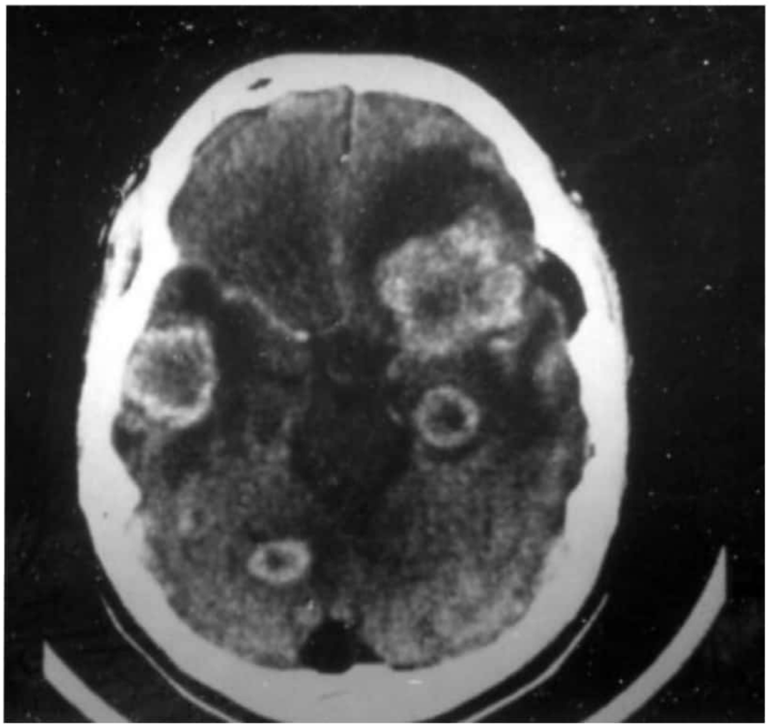
Progressive muscular atrophy is a rare form of motor neurone disease which only affects the lower motor neurones resulting in fasciculations and progressive wasting of upper and lower limbs.

A 47-year-old man with HIV disease presents to hospital with a tonic-clonic seizure.

He had initially presented six months previously with *Pneumocystis jirovecii* pneumonia with a CD4 T lymphocyte count of 10 cells/mm^3 and had subsequently started on highly active antiretroviral therapy. His most recent CD4 count, taken one month prior to his new presentation, was 50 cells/mm^3.

On examination he has no focal weakness, but both plantar responses are extensor. Fundoscopy is normal.

A CT scan of his brain is shown.



What is the diagnosis?

(Please select 1 option)

○ Primary CNS lymphoma

○ AIDS-related dementia

○ Cerebral toxoplasmosis

○ Progressive multifocal leukoencephalopathy

○ Brain abscess

Submit Answer

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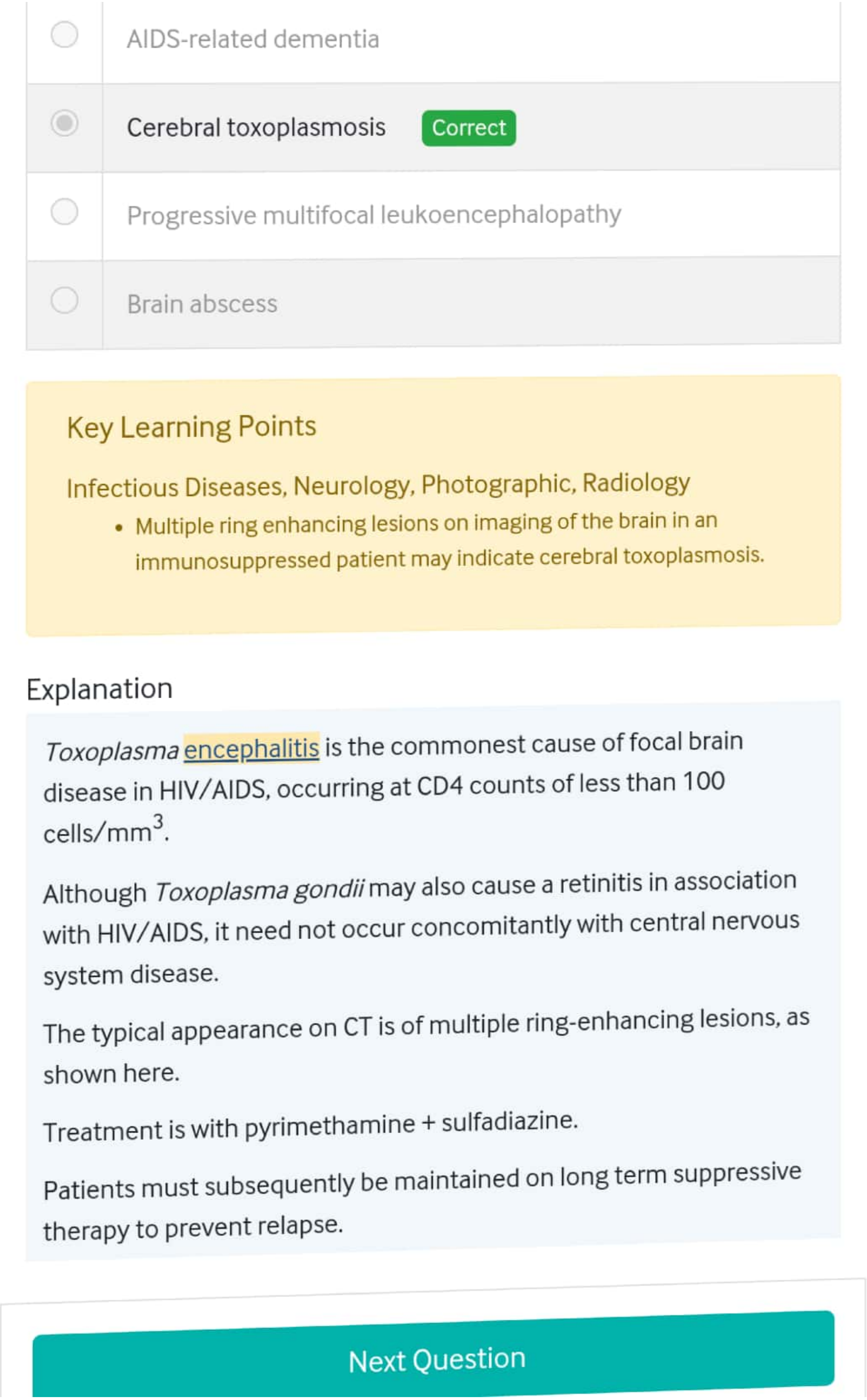

○ AIDS-related dementia

● Cerebral toxoplasmosis **Correct**

○ Progressive multifocal leukoencephalopathy

○ Brain abscess

Key Learning Points

Infectious Diseases, Neurology, Photographic, Radiology

- Multiple ring enhancing lesions on imaging of the brain in an immunosuppressed patient may indicate cerebral toxoplasmosis.

Explanation

Toxoplasma encephalitis is the commonest cause of focal brain disease in HIV/AIDS, occurring at CD4 counts of less than 100 cells/mm^3.

Although *Toxoplasma gondii* may also cause a retinitis in association with HIV/AIDS, it need not occur concomitantly with central nervous system disease.

The typical appearance on CT is of multiple ring-enhancing lesions, as shown here.

Treatment is with pyrimethamine + sulfadiazine.

Patients must subsequently be maintained on long term suppressive therapy to prevent relapse.

A 20-year-old woman with progressive unsteadiness was seen by her GP.

She had noticed a worsening unsteadiness over the past few months and in addition complained of some numbness and weakness of the lower legs. Her friends had also noticed that her speech had recently become slurred. She had no past medical history but admitted that she had always been 'clumsy' as a child. She was a vegan, and adopted.

On examination, she was alert and orientated and had a dysarthria. Fundoscopy revealed bilateral optic atrophy and horizontal nystagmus. The rest of the cranial nerves appeared normal.

Examination of the upper limb revealed normal tone, but depressed reflexes bilaterally. Power appeared normal; however there was reduced vibration sense, which was felt at the elbow. There was some hesitancy on finger to nose testing.

Examination of the lower limb revealed bilateral pes cavus with distal muscle wasting. Tone was reduced bilaterally and she had absent ankle and knee jerks with bilateral extensor plantar responses. There was a pyramidal distribution of weakness affecting both legs. Sensory examination showed reduced vibration and joint position sense felt at the hip bilaterally. Gait appeared ataxic and Romberg's test was positive.

Examination of the cardiovascular system revealed a double apical impulse and an ejection systolic murmur along the left sternal edge.

Blood tests showed:

Full blood count	Normal	-
Biochemical profile	Normal	-
Glucose	9.8 mmol/L	(3.5 - 5.5)
B12 and folate	Normal	-

Lumbar puncture revealed:

CSF protein	0.45 g/L	(0.15 - 0.45)
CSF WCC	3 per ml	(≤5)
CSF RCC	2 per ml	(≤5)
CSF oligoclonal bands	Absent	-

What is the likely diagnosis?

(Please select 1 option)

○ Subacute combined degeneration of the cord

○ Multiple sclerosis

○ Friedreich's ataxia

○ Vitamin A deficiency

○ Hereditary motor sensory neuropathy (Roussy-Levy variant)

Submit Answer

● Friedreich's ataxia **Correct**

○ Vitamin A deficiency

○ Hereditary motor sensory neuropathy (Roussy-Levy variant)

Key Learning Points

Neurology

- Friedreich's ataxia is an autosomal recessive trinucleotide repeat disorder leading to gross neurodegeneration.

Explanation

This patient presents with progressive gait ataxia, cerebellar dysfunction, pyramidal weakness and a sensory-motor neuropathy. In addition she has bilateral pes cavus and optic atrophy, elevated glucose level and cardiovascular findings in keeping with hypertrophic cardiomyopathy.

All these features are highly suggestive of Friedreich's ataxia, an autosomal recessive trinucleotide repeat disorder leading to neurodegeneration of dorsal root ganglia, cerebellum and corticospinal tracts.

Vitamin E deficiency can also present with similar features, and must be excluded.

Subacute combined degeneration of the cord leads to combined upper and lower motor signs with posterior column signs.

B12 and folate levels and full blood count are all normal in this patient.

Multiple sclerosis causes central demyelination and would not cause a peripheral neuropathy. In addition, positive oligoclonal bands are found in 90% of cases.

A 24-year-old woman presented to her GP with a six week history of double vision and painful right eye.

Three years ago she had had visual problems with her left eye and her symptoms on this occasion had lasted several days. There was no history of trauma or headache. She had no past medical history and was not taking any regular medication. Her mother had died of a neurological complaint. She was a smoker of 10 cigarettes per day and drank 21 units of alcohol per week.

On examination she was alert and orientated. On cranial nerve examination the right pupil appeared sluggish on reacting to light and visual acuity was 6/9 in the right and 6/6 in the left. There was no evidence of proptosis, or conjunctival injection. On testing ocular movements, she complained of diplopia on looking to the left and there was oculoparesis of the adduction of the right eye with nystagmus of the abducting eye. The rest of the cranial nerves were normal.

On examination of the peripheral nervous system, there was a bilateral past-pointing which seemed worse on the right. Lower limb examination revealed a mildly ataxic gait; but Romberg's test was negative. Tone, power, reflexes and sensation all appeared normal.

Investigations showed:

Haemoglobin	138 g/L	(130-180)
Mean cell volume	87 fL	(80-96)
White cell count	6.11×10^9/L	(4-11)
Platelets	198×10^9/L	(150-400)
Serum sodium	141 mmol/L	(137-144)
Serum potassium	4.3 mmol/L	(3.5-4.9)
Serum urea	6.1 mmol/L	(2.5-7.5)
Serum creatinine	87 µmol/L	(60-110)
Serum C-reactive protein	<5 U/L	(<5)
Fasting plasma glucose	5.6 mmol/L	(3-6)
Serum folate	9.5 µg/L	(2-11)
Serum B12	601 ng/L	(160-760)
Plasma TSH	3.02 mU/L	(0.5-5.0)
Plasma T4	67 nmol/L	(58-174)
Plasma T3	2.11 nmol/L	(1.07-3.18)

A lumbar puncture was performed and yielded the following data:

Opening pressure	11 cmH_2O	-
CSF Protein	0.56 g/L	-
CSF White cell count	3 cells per ml	(<5)
CSF red cell count	2 cells per ml	(<5)
CSF oligoclonal bands	Positive	-
Serum oligoclonal bands	Negative	-

Given the history and clinical findings what is the most likely diagnosis in this patient?

(Please select 1 option)

- ○ Friedreich's ataxia
- ○ Wernicke's encephalopathy
- ○ Multiple sclerosis
- ○ Brain stem glioma
- ○ Carotid-cavernous fistula

Submit Answer

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● Multiple sclerosis **Correct**

○ Brain stem glioma

○ Carotid-cavernous fistula

Key Learning Points

Neurology

- Young female patients with multiple cases of neuritis is suggestive of multiple sclerosis.

Explanation

This patient presents with a probable right optic neuritis and right internuclear ophthalmoplegia. In addition, she has peripheral features of subacute cerebellar syndrome.

Given that she has normal blood tests, positive oligoclonal bands in the cerebrospinal fluid and a previous history of probable optic neuritis, the likely cause in this young patient is multiple sclerosis.

A carotid-cavernous fistula would cause acute painful, pulsating exophthalmos, in addition to visual loss and diplopia.

Wernicke's encephalopathy is due to a nutritional deficiency of thiamine, presenting with a triad of acute mental confusion, ataxia with positive Romberg's test and ophthalmoplegia. Oculomotor findings include weakness of abduction, gaze-evoked nystagmus and internuclear ophthalmoplegia.

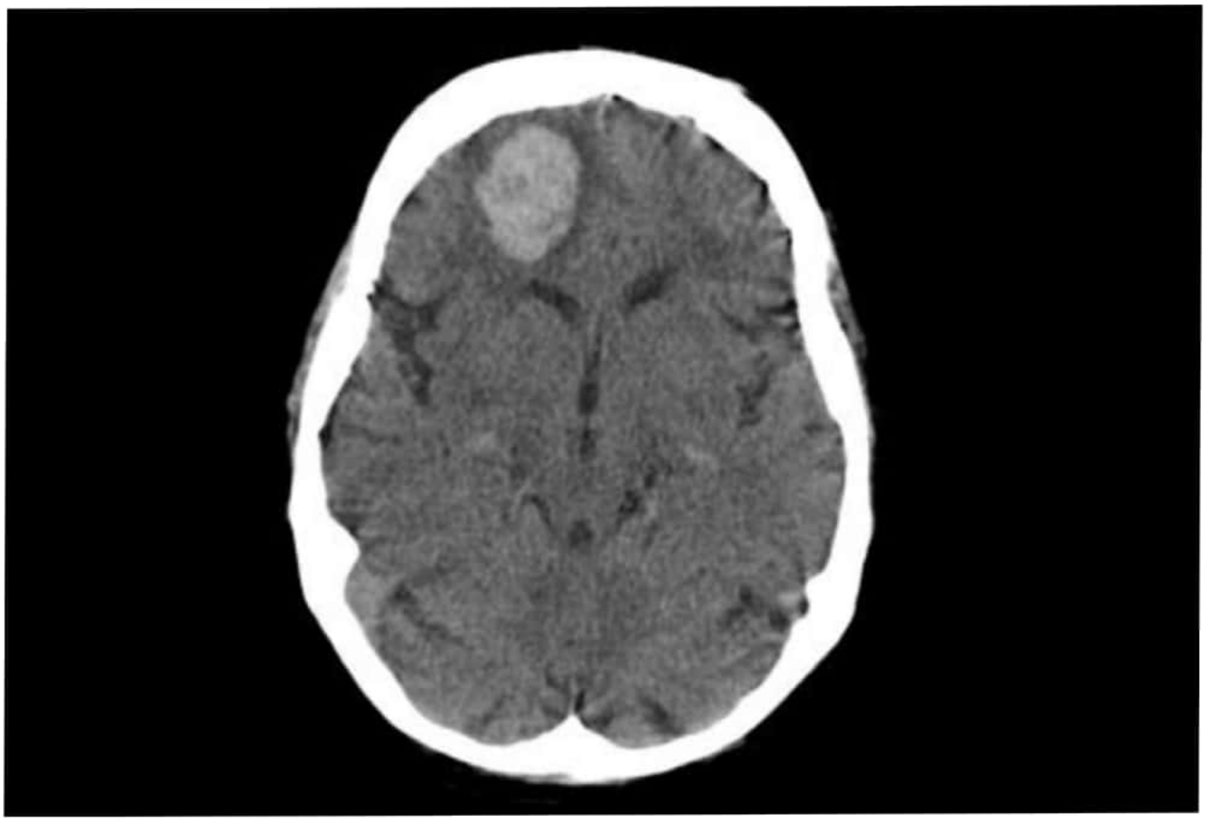
Freidreich's ataxia is an autosomal recessive trinucleotide repeat disorder, leading to neurodegeneration of dorsal root ganglia, cerebellum and corticospinal tracts.

Next Question

A 65-year-old woman presents with severe headache and confusion. She has a past medical history of atrial fibrillation and is taking warfarin.

On examination she has a temperature of 37.6℃ and neurological examination reveals a GCS of 13 with mild left-sided weakness in an upper motor neurone pattern and a left-sided upgoing plantar. A CT of the head is performed and one of the sections is shown below.

What is the most likely diagnosis?



(Please select 1 option)

○ Extradural haematoma

○ Intracranial right brain haematoma

○ Cerebral abscess

○ Acute anterior cerebral artery infarct

○ Subdural haemorrhage

○ Extradural haematoma

● Intracranial right brain haematoma **Correct**

○ Cerebral abscess

○ Acute anterior cerebral artery infarct

○ Subdural haemorrhage

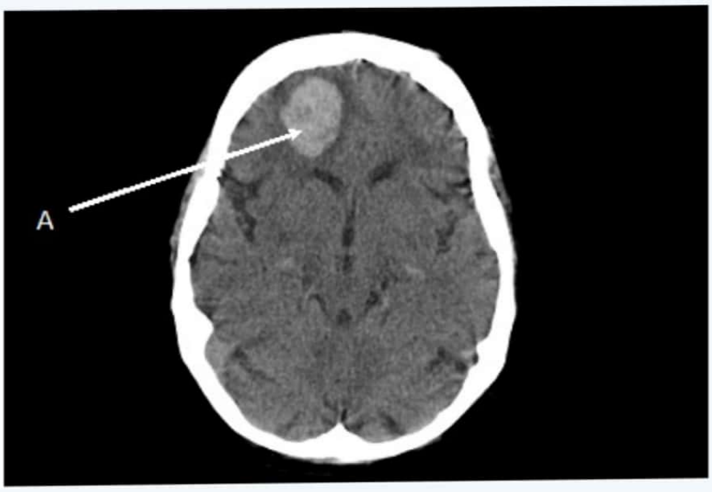
Key Learning Points

Neurology

- a well circumscribed high attenuation lesion with some surrounding oedema on a non contrast CT is consistent with an intracranial haematoma.

Explanation

This is a non-contrast CT head which demonstrates a well circumscribed high attenuation lesion with some surrounding oedema in the frontal lobe (A) consistent with an intracranial haematoma.



Acute cerebral infarcts are typically hypodense with vasogenic oedema, they conform to arterial blood supply territories and are typically wedge-shaped (when not lacunar).

Cerebral abscesses are usually hypodense with surrounding oedema, peripheral ring enhancement is characteristically seen when contrast is administered.

Acute extradural and subdural haematomas would both be high attenuation however both are anatomically located next to the skull - extradural haematomas have a convex border whilst subdural haematomas have a concave border. Acutely blood is high attenuation (bright) on CT, as time passes the degree of attenuation decreases and the area eventually becomes hypodense. Care must be taken not to miss bleeds when they are isodense with cerebral tissue.

Next Question

A 51-year-old man was referred to the acute medical intake with a two week history of severe pain around his right eye.

The pain had started without any apparent precipitating event and had woken him from his sleep. He described the pain as severe and 'boring' and radiated upwards over the right frontal and temporal region. The pain was not constant, but seemed to start every evening and persist for periods of 20 minutes to over an hour. The headaches were associated with watering of his right eye and a blocked left nostril. There was no past history of similar episodes and no other past history of note.

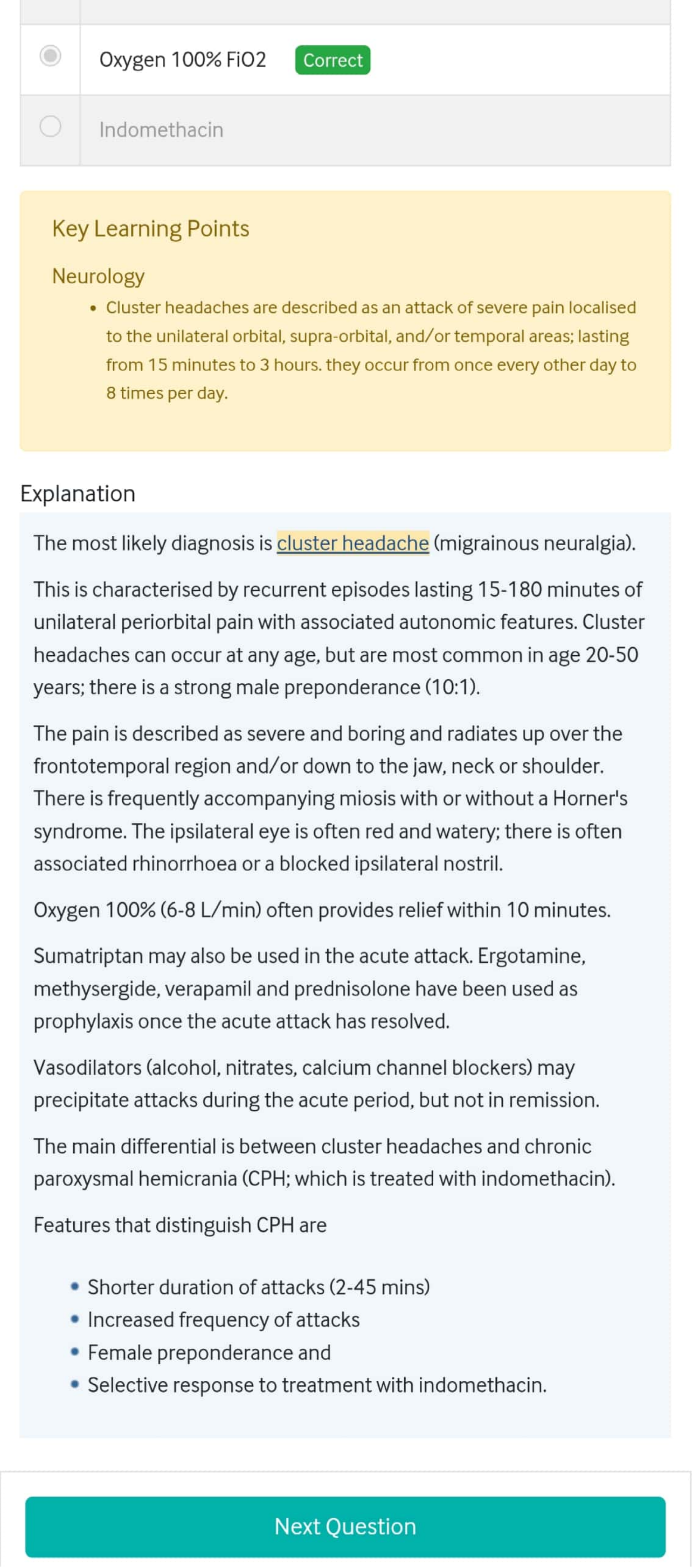
On examination he was alert and orientated with a Glasgow coma scale score of 15/15. His blood pressure was 125/75 mmHg. He was afebrile and there was no neck stiffness. The right eye was red with conjunctival injection and mild oedema of the eyelid. There was a partial right sided ptosis and miosis.

Which of the following therapeutic options is most likely to alleviate his symptoms?

(Please select 1 option)

○	Carbamazepine
○	Verapamil
○	Aspirin
○	Oxygen 100% FiO2
○	Indomethacin

Submit Answer



●	Oxygen 100% FiO2 **Correct**
○	Indomethacin

Key Learning Points

Neurology

- Cluster headaches are described as an attack of severe pain localised to the unilateral orbital, supra-orbital, and/or temporal areas; lasting from 15 minutes to 3 hours. they occur from once every other day to 8 times per day.

Explanation

The most likely diagnosis is cluster headache (migrainous neuralgia).

This is characterised by recurrent episodes lasting 15-180 minutes of unilateral periorbital pain with associated autonomic features. Cluster headaches can occur at any age, but are most common in age 20-50 years; there is a strong male preponderance (10:1).

The pain is described as severe and boring and radiates up over the frontotemporal region and/or down to the jaw, neck or shoulder. There is frequently accompanying miosis with or without a Horner's syndrome. The ipsilateral eye is often red and watery; there is often associated rhinorrhoea or a blocked ipsilateral nostril.

Oxygen 100% (6-8 L/min) often provides relief within 10 minutes.

Sumatriptan may also be used in the acute attack. Ergotamine, methysergide, verapamil and prednisolone have been used as prophylaxis once the acute attack has resolved.

Vasodilators (alcohol, nitrates, calcium channel blockers) may precipitate attacks during the acute period, but not in remission.

The main differential is between cluster headaches and chronic paroxysmal hemicrania (CPH; which is treated with indomethacin).

Features that distinguish CPH are

- Shorter duration of attacks (2-45 mins)
- Increased frequency of attacks
- Female preponderance and
- Selective response to treatment with indomethacin.

‹ ›

A 56-year-old man with a history of hypertension, hiatus hernia, and occasional cocaine use presents to the Emergency department with acute onset of dizziness, chest pain and bilateral limb weakness.

On the morning of admission, he experienced an acute onset of chest discomfort associated with dizziness and had to sit down in a chair. Shortly afterwards, he noticed tingling in all extremities, became diaphoretic, and could not move his limbs. He denies involvement of his face, vertigo, or diplopia. His speech is clear. The patient denies any trauma.

On examination, he is apyrexial, wearing a collar, tachypnoiec and gasping for air between short phrases. He is weak in all his limbs (right more than left) with a sensory loss to the level of C3/4. Sensation was absent to pin-prick and temperature up to C3/4 level. Vibration sense and proprioception were normal. Deep tendon reflexes were absent throughout with decreased anal tone. Plantars were upgoing bilaterally.

Investigations showed:

Hb	129 g/L	(130-180)
WCC	5.7 ×10^9/L	(4-11)
Platelets	250 ×10^9/L	(150-400)
Plasma sodium	145 mmol/L	(137-144)
Plasma potassium	3.9 mmol/L	(3.5-4.9)
Plasma urea	6.8 mmol/L	(2.5-7.5)
Plasma creatinine	101 μmol/L	(60-110)
Plasma glucose	5.9 mmol/L	(3.0-6.0)
CSF opening pressure	16 mm H_2O	(50-180)
CSF cell count	<2 ml^{-1}	(≤5)
CSF protein	0.4 g/L	(0.15-0.45)
CSF glucose	6.8 mmol/L	(3.3-4.4)
CSF microbiology	No organism on Gram stain or growth on culture	
Urine toxicology	Positive for cocaine only	
CT scan of brain	Normal	

What is the likely diagnosis?

(Please select 1 option)

○ Spinal cord ischaemic stroke at C3/4

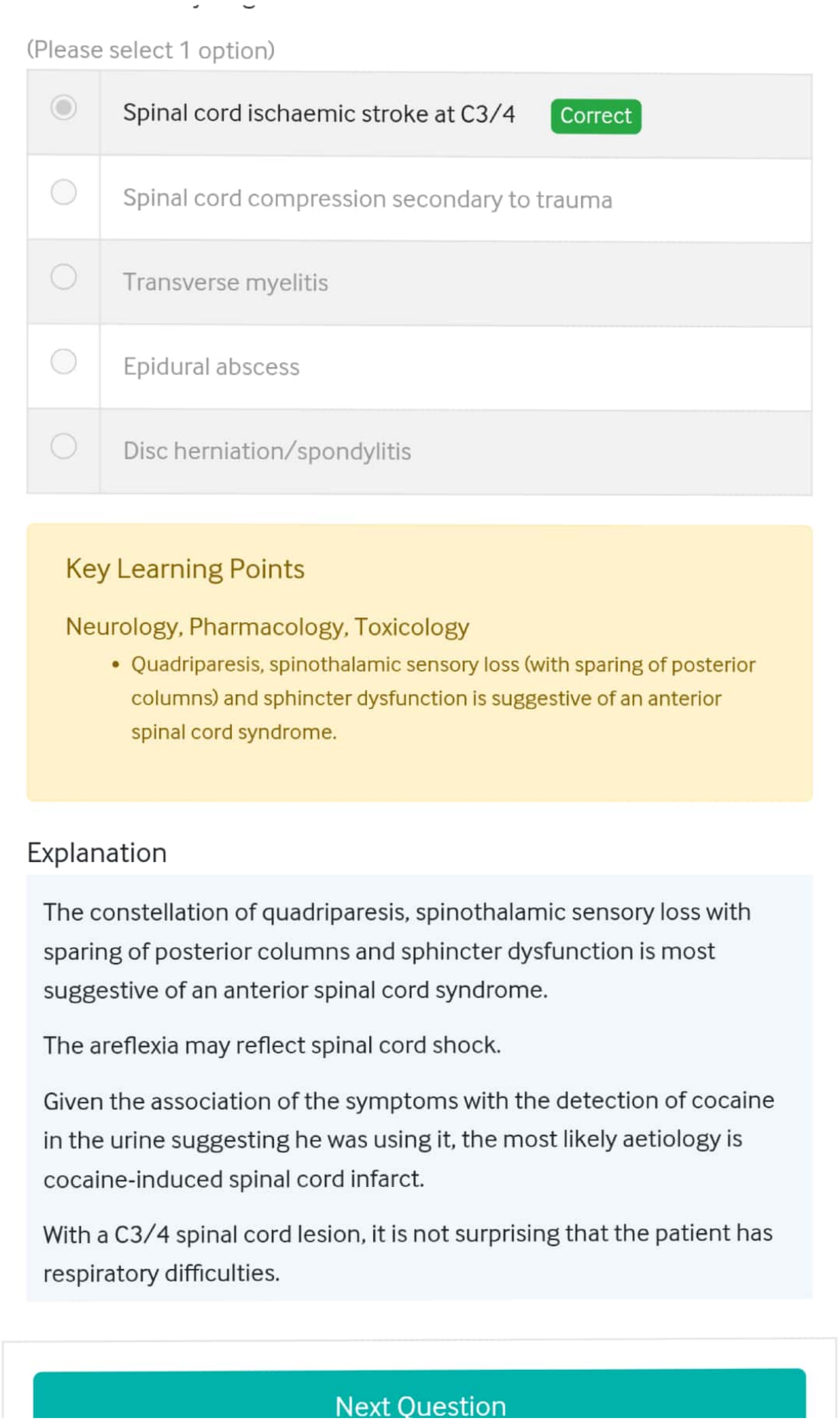
○ Spinal cord compression secondary to trauma

○ Transverse myelitis

○ Epidural abscess

○ Disc herniation/spondylitis

Submit Answer



(Please select 1 option)

- ● Spinal cord ischaemic stroke at C3/4 **Correct**
- ○ Spinal cord compression secondary to trauma
- ○ Transverse myelitis
- ○ Epidural abscess
- ○ Disc herniation/spondylitis

Key Learning Points

Neurology, Pharmacology, Toxicology

- Quadriparesis, spinothalamic sensory loss (with sparing of posterior columns) and sphincter dysfunction is suggestive of an anterior spinal cord syndrome.

Explanation

The constellation of quadriparesis, spinothalamic sensory loss with sparing of posterior columns and sphincter dysfunction is most suggestive of an anterior spinal cord syndrome.

The areflexia may reflect spinal cord shock.

Given the association of the symptoms with the detection of cocaine in the urine suggesting he was using it, the most likely aetiology is cocaine-induced spinal cord infarct.

With a C3/4 spinal cord lesion, it is not surprising that the patient has respiratory difficulties.

Question 107

Question 107 of 129

<   >

A 29-year-old man presents with a six month history of nasal congestion. He was seen by an ENT consultant who diagnosed sinusitis. He presents now with a two day history of right periorbital swelling and diplopia.

Examination reveals him to be unwell, although he has no neck stiffness or photophobia; his temperature is 37.4°C. He has marked drooping of the right eyelid with the right eye congested and deviated to the right with an enlarged right pupil. Left eye appears normal. Fundoscopy is normal. There is also loss of sensation of the right forehead.

What is the likely diagnosis?

(Please select 1 option)

- ○ Cavernous sinus thrombosis
- ○ Meningitis
- ○ Tolosa-Hunt syndrome
- ○ Pituitary adenoma
- ○ Epidural abscess

Submit Answer

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(Please select 1 option)

● Cavernous sinus thrombosis Correct

○ Meningitis

○ Tolosa-Hunt syndrome

○ Pituitary adenoma

○ Epidural abscess

Key Learning Points

Emergency Medicine, Neurology, Ophthalmology

- Intermittent headaches with neurology affecting multiple cranial nerves is suggestive of Cavernous Sinus Thrombosis.

Explanation

The history is typical for cavernous sinus thrombosis possibly secondary to sinusitis. This is a life threatening condition.

Urgent CT brain and antibiotics are needed.

The structures within and beside the cavernous sinus and their relative position can be memorised with the following mnemonic:

O TOM CAT

O TOM are the first letters of structures of the lateral wall of the cavernous sinus arranged vertically, from the top to the bottom. CA are the first letters of the structures located within the sinus.

O: Oculomotor nerve

T: Trochlear nerve

O: Ophthalmic branch of trigeminal nerve

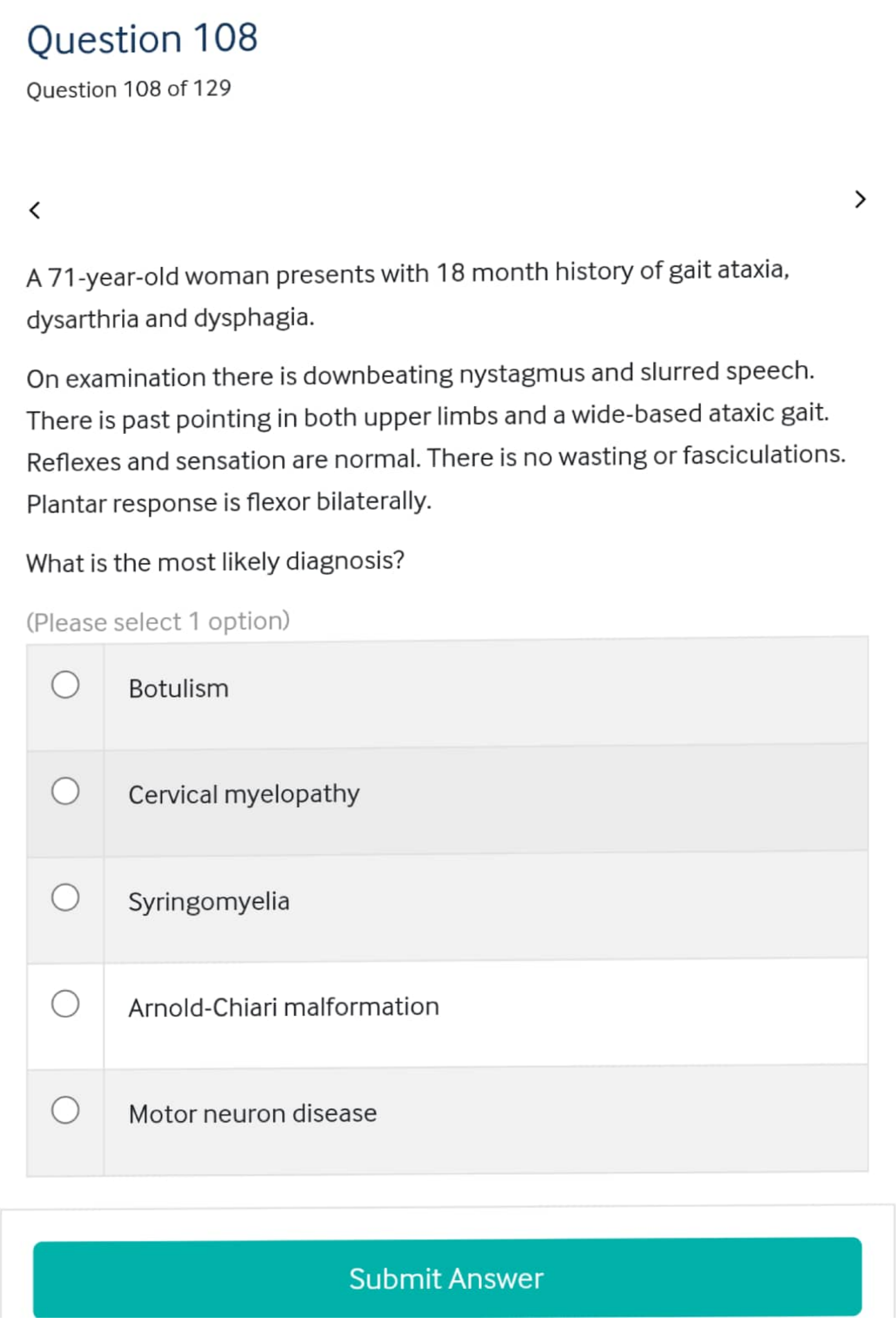
M: Maxillary branch of trigeminal nerve

C: Internal carotid artery

A: Abducent nerve

T: Trochlear nerve

Next Question



Question 108

Question 108 of 129

A 71-year-old woman presents with 18 month history of gait ataxia, dysarthria and dysphagia.

On examination there is downbeating nystagmus and slurred speech. There is past pointing in both upper limbs and a wide-based ataxic gait. Reflexes and sensation are normal. There is no wasting or fasciculations. Plantar response is flexor bilaterally.

What is the most likely diagnosis?

(Please select 1 option)

○ Botulism

○ Cervical myelopathy

○ Syringomyelia

○ Arnold-Chiari malformation

○ Motor neuron disease

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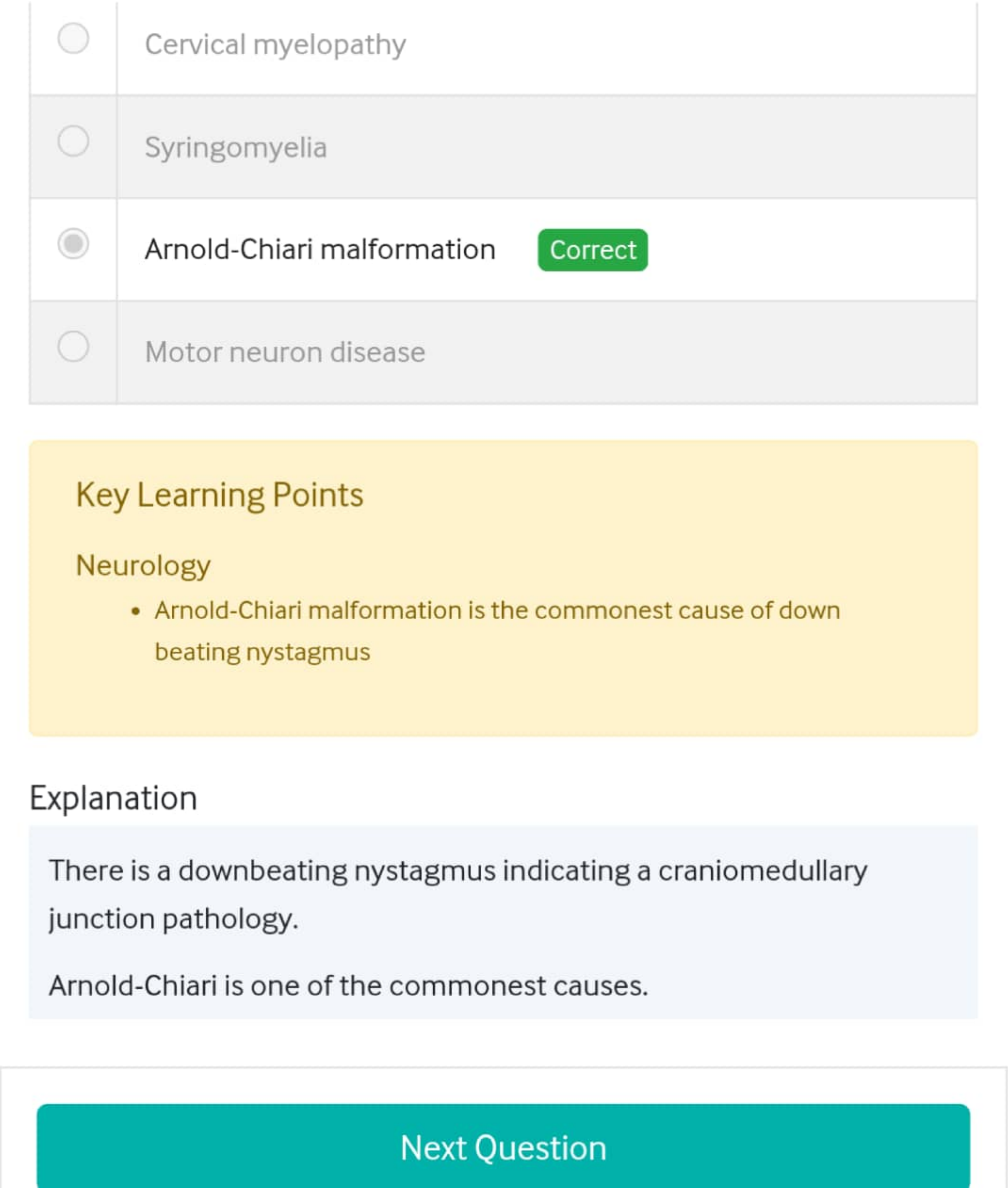

○ Cervical myelopathy

○ Syringomyelia

● Arnold-Chiari malformation Correct

○ Motor neuron disease

Key Learning Points

Neurology

- Arnold-Chiari malformation is the commonest cause of down beating nystagmus

Explanation

There is a downbeating nystagmus indicating a craniomedullary junction pathology.

Arnold-Chiari is one of the commonest causes.

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Question 109

Question 109 of 129

You are referred a 40-year-old woman in a psychiatry ward who has a long history of schizophrenia resulting in multiple hospitalisations in the past.

She gives a several week history of feeling generally unwell associated with increasing stiffness, affecting the jaws and arms. Over the last few years she has been started on haloperidol with good symptom control. Her temperature was 38.5°C and BP 175/85 mmHg.

Which of the following drug treatments would you consider for this patient's condition?

(Please select 1 option)

- ○ Benzatropine
- ○ Dantrolene
- ○ Cefotaxime
- ○ Prednisolone
- ○ Lithium

Submit Answer

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● Dantrolene **Correct**

○ Cefotaxime

○ Prednisolone

○ Lithium

Key Learning Points

Neurology, Therapeutics

- Concomitant treatment with lithium or anticholinergics may increase the risk of NMS. It is manifested by fever, rigidity, altered mental status and autonomic dysfunction. Treatment includes withdrawal of the offending agent, reduction of body temperature with antipyretics. Dantrolene, bromocriptine or levodopa preparations may be beneficial.

Explanation

The diagnosis is neuroleptic malignant syndrome (NMS) which can occur at any time during the treatment of antipsychotic medications. Concomitant treatment with lithium or anticholinergics may increase the risk of NMS. It is manifested by fever, rigidity, altered mental status and autonomic dysfunction. Treatment includes withdrawal of the offending agent, reduction of body temperature with antipyretics. Dantrolene, bromocriptine or levodopa preparations may be beneficial.

Serotonin syndrome is a differential diagnosis for NMS but the two can be easily distinguished on thorough history and examination.

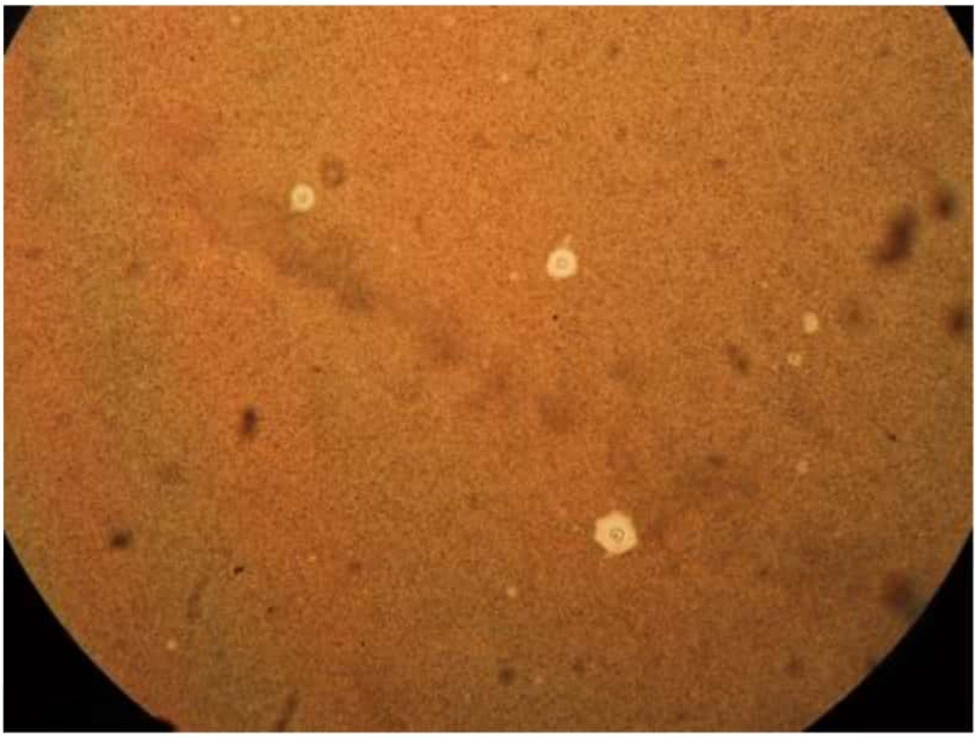
NMS develops over days and weeks, whereas serotonin syndrome can develop over 24 hours. Serotonin syndrome develops neuromuscular hyperreactivity, (myoclonus, tremors, hyperreflexia) whereas NMS involves sluggish neuromuscular response (bradyreflexia, rigidity). Hyperreflexia and myoclonus are rare in NMS. Also resolution of NMS takes up to nine days whereas (usually) it takes less than 24 hours in serotonin syndrome.

Hyperthermia, muscle rigidity, leukocytosis, elevated CK, altered hepatic function and metabolic acidosis are common to severe cases in both conditions, which is why a thorough history and physical examination are essential in order to differentiate between the two.

Next Question

A 47-year-old man with advanced HIV disease presented with fever and obtundation. He has not attended his clinic appointments for several years and is not taking antiretroviral therapy.

A CT scan of his brain was normal. A lumbar puncture was performed; microscopy of his cerbrospinal fluid is shown below.



What treatment should this patient receive?

(Please select 1 option)

○	High-dose intravenous aciclovir
○	Amphotericin B + flucytosine
○	Highly active antiretroviral therapy
○	Sulfadiazine + pyrimethamine
○	High-dose intravenous cefotaxime

Submit Answer

(Please select 1 option)

○	High-dose intravenous aciclovir
●	Amphotericin B + flucytosine **Correct**
○	Highly active antiretroviral therapy
○	Sulfadiazine + pyrimethamine
○	High-dose intravenous cefotaxime

Key Learning Points

Infectious Diseases, Neurology, Photographic

- Cryptococcal meningitis is an AIDS defining illness

Explanation

The slide is an India ink stain of CSF that shows typical yeast-like forms of *Cryptococcus* sp; the thick polysaccharide capsule is highlighted against the darker backgound of black ink.

The history is characteristic of cryptococcal disease in AIDS.

Treatment is with amphotericin B and flucytosine (5FC); patients then require lifetime suppression with fluconazole.

Next Question

A 17-year-old girl presented with a two day history of severe back pain.

A plain x ray film of her spine is normal. Two days later, she complained of tingling in her fingers and toes. The next day she became generally weak. She presented in the emergency department.

On examination, she has bilateral lower motor neurone facial weakness, tetraparesis with weakness in all limbs (4/5), areflexia, flexor plantars and normal sensation.

A lumbar puncture was performed and the CSF analysis showed:

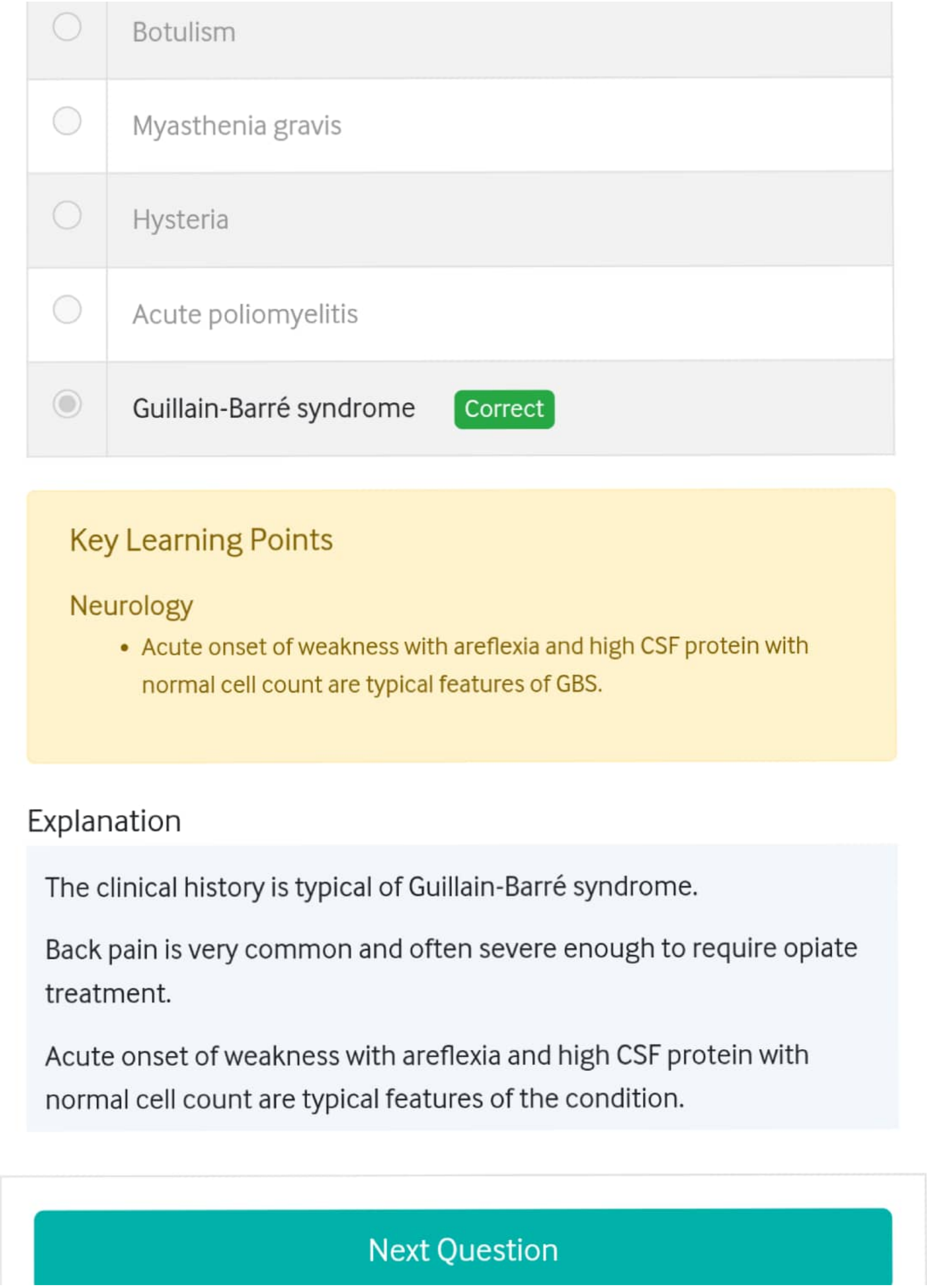
Protein	1.4 g/L	(<0.4)
Glucose	Normal	-
Cells	0	(<5 ml^{-1})

What is the most likely diagnosis?

(Please select 1 option)

○	Botulism
○	Myasthenia gravis
○	Hysteria
○	Acute poliomyelitis
○	Guillain-Barré syndrome

Submit Answer



○ Botulism

○ Myasthenia gravis

○ Hysteria

○ Acute poliomyelitis

● Guillain-Barré syndrome **Correct**

Key Learning Points

Neurology

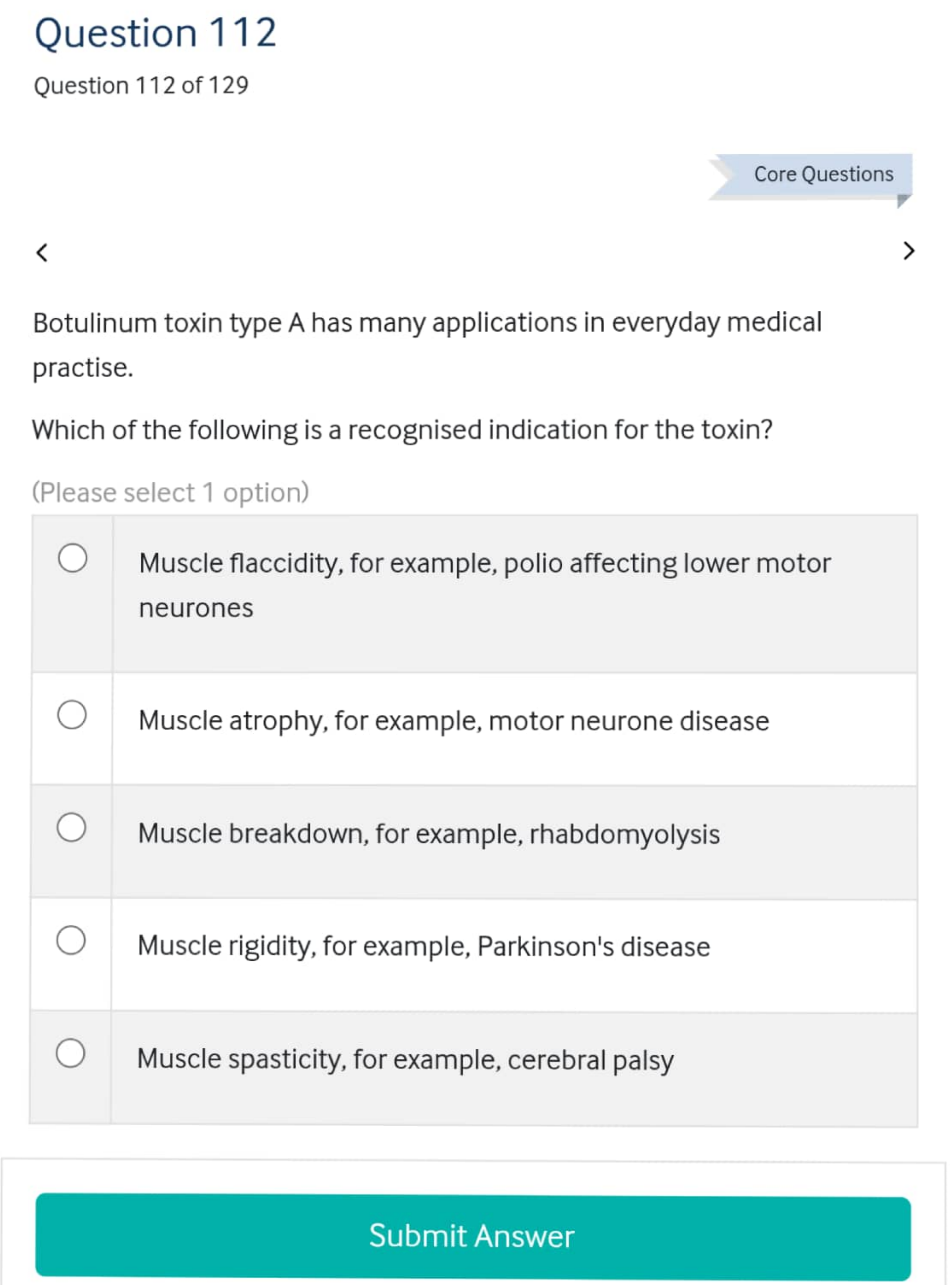
- Acute onset of weakness with areflexia and high CSF protein with normal cell count are typical features of GBS.

Explanation

The clinical history is typical of Guillain-Barré syndrome.

Back pain is very common and often severe enough to require opiate treatment.

Acute onset of weakness with areflexia and high CSF protein with normal cell count are typical features of the condition.



Question 112

Question 112 of 129

Core Questions

Botulinum toxin type A has many applications in everyday medical practise.

Which of the following is a recognised indication for the toxin?

(Please select 1 option)

○ Muscle flaccidity, for example, polio affecting lower motor neurones

○ Muscle atrophy, for example, motor neurone disease

○ Muscle breakdown, for example, rhabdomyolysis

○ Muscle rigidity, for example, Parkinson's disease

○ Muscle spasticity, for example, cerebral palsy

○ Muscle spasticity, for example, cerebral palsy Correct

Key Learning Points

Dermatology, Neurology, Pharmacology, Therapeutics

- Botulinum toxin type A (or trade name Botox®) acts by decreasing transmission of acetylcholine across the neuromuscular junction and can induce temporary flaccid paralysis.

Explanation

Botulinum toxin type A (or trade name Botox®) acts by decreasing transmission of acetylcholine across the neuromuscular junction in striated muscles, so inducing a temporary flaccid paralysis in the muscle into which it is injected.

Wrist and hand disability due to upper limb spasticity associated with stroke in adults, and use in cases of muscle spasticity, such as cases of cerebral palsy are well recognised indications for the use of Botox®.

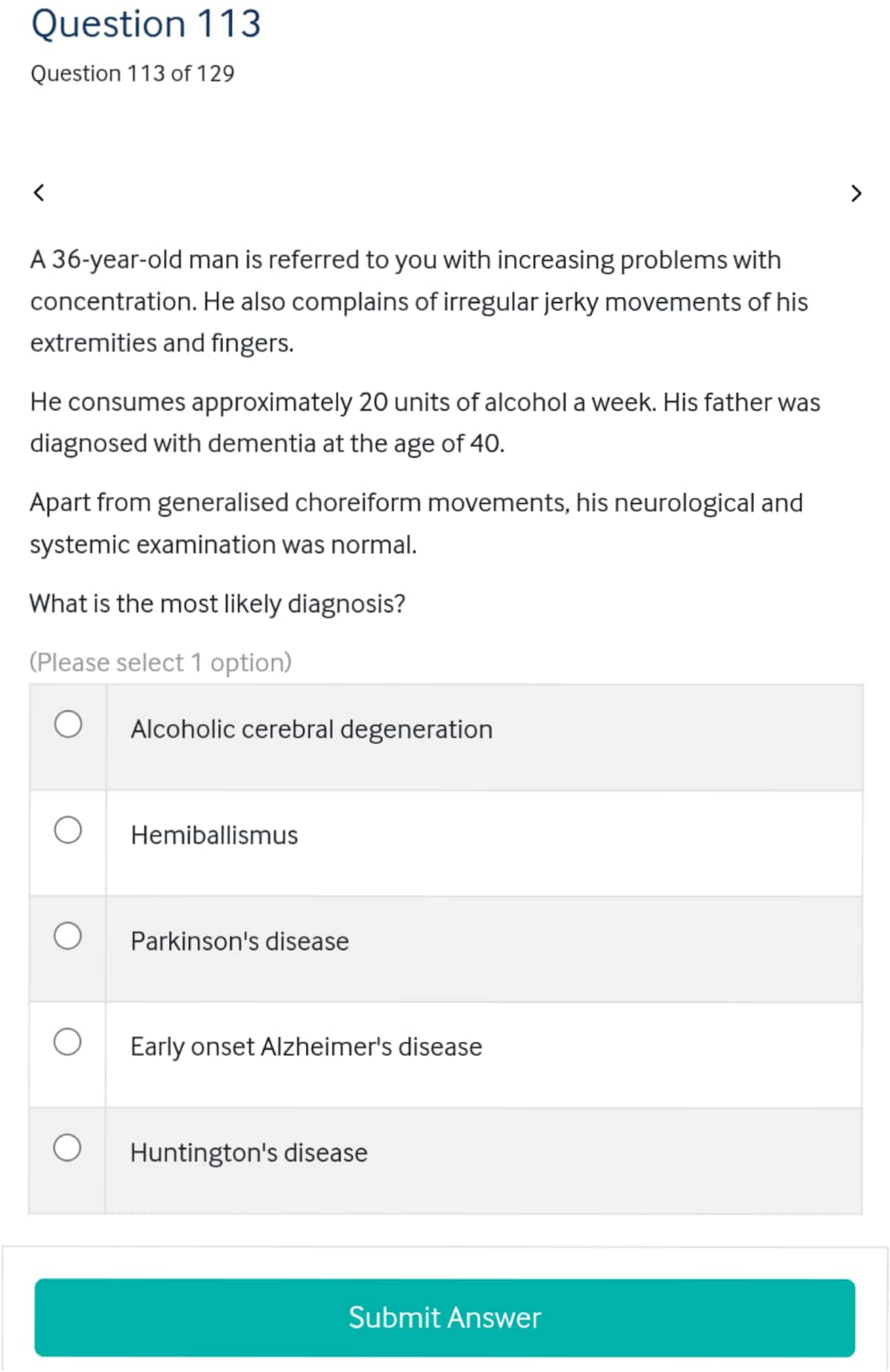
Though not licensed, Botox has been used in other medical conditions, including:

- Primary axillary hyperhidrosis
- Blepharospasm
- Strabismus, and
- Cervical dystonia.

Reference:

Electronic Medicines Compendium (eMC). Botox 100 Units.

Next Question



Question 113

Question 113 of 129

A 36-year-old man is referred to you with increasing problems with concentration. He also complains of irregular jerky movements of his extremities and fingers.

He consumes approximately 20 units of alcohol a week. His father was diagnosed with dementia at the age of 40.

Apart from generalised choreiform movements, his neurological and systemic examination was normal.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Alcoholic cerebral degeneration
- ○ Hemiballismus
- ○ Parkinson's disease
- ○ Early onset Alzheimer's disease
- ○ Huntington's disease

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○ Early onset Alzheimer's disease

● Huntington's disease Correct

Key Learning Points

Genetics, Neurology

- Huntington's disease is an autosomally inherited condition due to an expanded CAG trinucleotide repeat on the short arm of chromosome 4. It is characterised by progressive dementia and worsening choreiform movements. Symptoms typically appear between 30 and 50 years of age.

Explanation

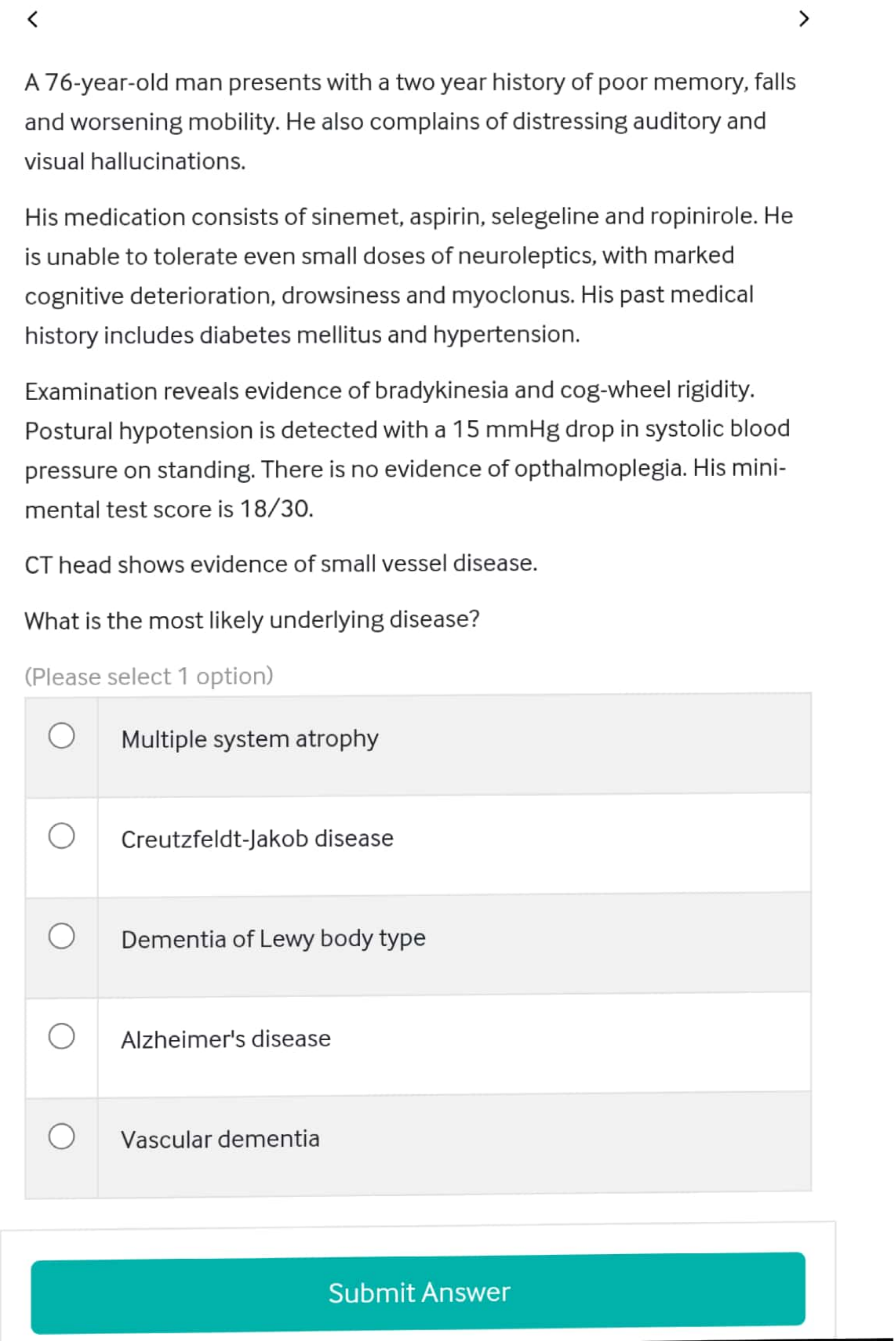
Huntington's disease is an autosomally inherited condition due to an expanded CAG trinucleotide repeat on the short arm of chromosome 4. It is characterised by progressive dementia and worsening choreiform movements.

Symptoms typically appear between 30 and 50 years of age.

Genetic testing now provides an accurate method of establishing the diagnosis.

Average life span after clinical onset is about 15 years.

Next Question



A 76-year-old man presents with a two year history of poor memory, falls and worsening mobility. He also complains of distressing auditory and visual hallucinations.

His medication consists of sinemet, aspirin, selegeline and ropinirole. He is unable to tolerate even small doses of neuroleptics, with marked cognitive deterioration, drowsiness and myoclonus. His past medical history includes diabetes mellitus and hypertension.

Examination reveals evidence of bradykinesia and cog-wheel rigidity. Postural hypotension is detected with a 15 mmHg drop in systolic blood pressure on standing. There is no evidence of opthalmoplegia. His mini-mental test score is 18/30.

CT head shows evidence of small vessel disease.

What is the most likely underlying disease?

(Please select 1 option)

- ○ Multiple system atrophy
- ○ Creutzfeldt-Jakob disease
- ○ Dementia of Lewy body type
- ○ Alzheimer's disease
- ○ Vascular dementia

● Dementia of Lewy body type **Correct**

○ Alzheimer's disease

○ Vascular dementia

Key Learning Points

Neurology

- Characteristic features of Lewy body dementia

Explanation

Dementia of Lewy body type accounts for 10-20% of cases of dementia.

Lewy bodies are intraneuronal, eosinophilic inclusion bodies found in cortical areas (they are also found in brainstem of patients with Parkinson's disease). The main clues to the diagnosis are the characteristic severe neuroleptic sensitivity reactions, and the presence of hallucinations (and delusions).

Alzheimer's disease (AD) is less likely in view of presence of vascular risk factors. Hallucinations and delusions are also less likely in AD.

The postural hypotension is a red herring for autonomic dystrophy (intellect spared in Multiple system atrophy) as it is more likely to be iatrogenic.

Creutzfeldt-Jakob disease in older people is rapidly fatal within a few months characterised by progressive multifocal neurological dysfunction (ataxia, pyramidal/extrapyramidal signs, stroke, cerebellar syndrome) and dementia.

Next Question

A 28-year-old man was admitted to the Emergency department after attending his weekly judo club meeting.

He had felt well and had been participating actively. Within a few minutes of his final fight, he developed pain in his neck, vertigo, blurred vision and diplopia. He had difficulty in balancing himself with a tendency to fall to the right and incoordination of right upper and lower limbs.

There was accompanying facial asymmetry, decreased hearing from right side, difficulty in swallowing, and associated weakness of right side of body.

There was no past medical history of note and he was a non-smoker.

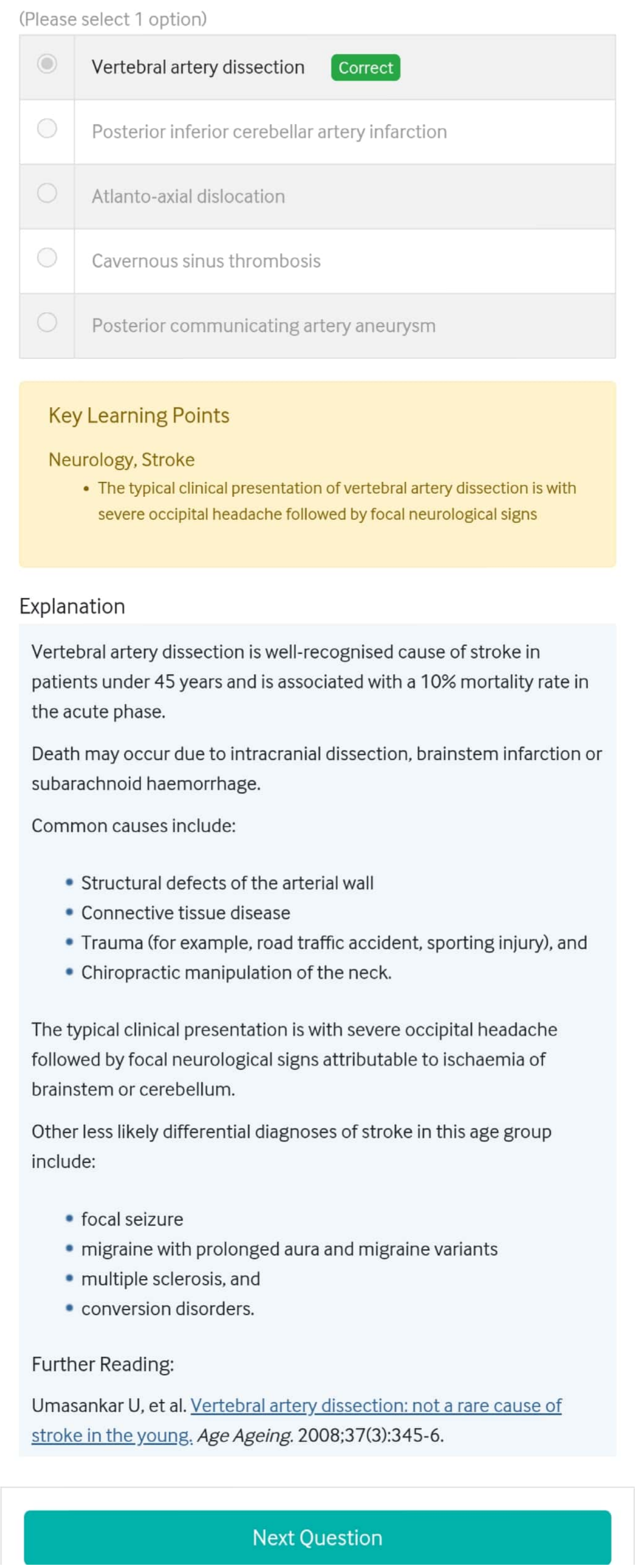
On examination, a right sided Horner's syndrome was noted. There was nystagmus (fast phase to the right), sensorineural hearing loss in the right ear and paresis of soft palate on right side. There was reduced tone and power in upper and lower limbs on the right. Deep tendon reflexes were brisk on the right side and plantar response was extensor.

Sensory examination revealed a crossed hemianaesthesia with involvement of the face on the right side.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Vertebral artery dissection
- ○ Posterior inferior cerebellar artery infarction
- ○ Atlanto-axial dislocation
- ○ Cavernous sinus thrombosis
- ○ Posterior communicating artery aneurysm



(Please select 1 option)

● Vertebral artery dissection Correct

○ Posterior inferior cerebellar artery infarction

○ Atlanto-axial dislocation

○ Cavernous sinus thrombosis

○ Posterior communicating artery aneurysm

Key Learning Points

Neurology, Stroke

- The typical clinical presentation of vertebral artery dissection is with severe occipital headache followed by focal neurological signs

Explanation

Vertebral artery dissection is well-recognised cause of stroke in patients under 45 years and is associated with a 10% mortality rate in the acute phase.

Death may occur due to intracranial dissection, brainstem infarction or subarachnoid haemorrhage.

Common causes include:

- Structural defects of the arterial wall
- Connective tissue disease
- Trauma (for example, road traffic accident, sporting injury), and
- Chiropractic manipulation of the neck.

The typical clinical presentation is with severe occipital headache followed by focal neurological signs attributable to ischaemia of brainstem or cerebellum.

Other less likely differential diagnoses of stroke in this age group include:

- focal seizure
- migraine with prolonged aura and migraine variants
- multiple sclerosis, and
- conversion disorders.

Further Reading:

Umasankar U, et al. Vertebral artery dissection: not a rare cause of stroke in the young. *Age Ageing.* 2008;37(3):345-6.

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A 66-year-old woman was seen by her GP with a history of sudden onset right visual disturbance associated with right temporal throbbing headache. She described the visual disturbance as a moving silhouette descending from above leading to complete visual loss. This lasted approximately one minute and resolved.

In addition she complained of pain when combing her hair and also pain in her jaw when chewing food. This had happened several times in the last week. She had a history of polymyalgia rheumatica and hypertension. Her regular medication was bendroflumethiazide 2.5 mg/day.

On examination she was alert and orientated. Her blood pressure was 140/75 mmHg and pulse was 76/min and regular. She was apyrexial. On cardiovascular examination she had an ejection systolic murmur radiating into the neck and no carotid bruits.

Fundoscopy examination revealed bilateral silver wiring and AV nipping. Pupils were equal and reactive and the rest of the cranial nerves were normal. The right temporal/jaw area appeared tender and pulsatile. Peripheral nervous system examination was normal.

Investigations showed:

Haemoglobin	106 g/L	(115-165)
White cell count	5.6 ×10^9/L	(4-11)
Platelets	500 ×10^9/L	(150-400)
Mean cell volume	86 fL	(80-96)
ESR (Westergren)	100 mm/1st hr	(0-30)
Cholesterol	6.2 mmol/L	(<5.2)
Sodium	134 mmol/L	(137-144)
Potassium	4.0 mmol/L	(3.5-4.9)
Urea	5.6 mmol/L	(2.5-7.5)
Creatinine	120 µmol/L	(60-110)
Glucose	5.7 mmol/L	(3.0-6.0)
C reactive protein	5 mg/L	(<10)

Carotid Dopplers:

Right internal carotid artery	<50% stenosis
Left internal carotid artery	100% stenosis

What is the diagnosis?

(Please select 1 option)

○ Septic embolic stroke affecting right ophthalmic artery

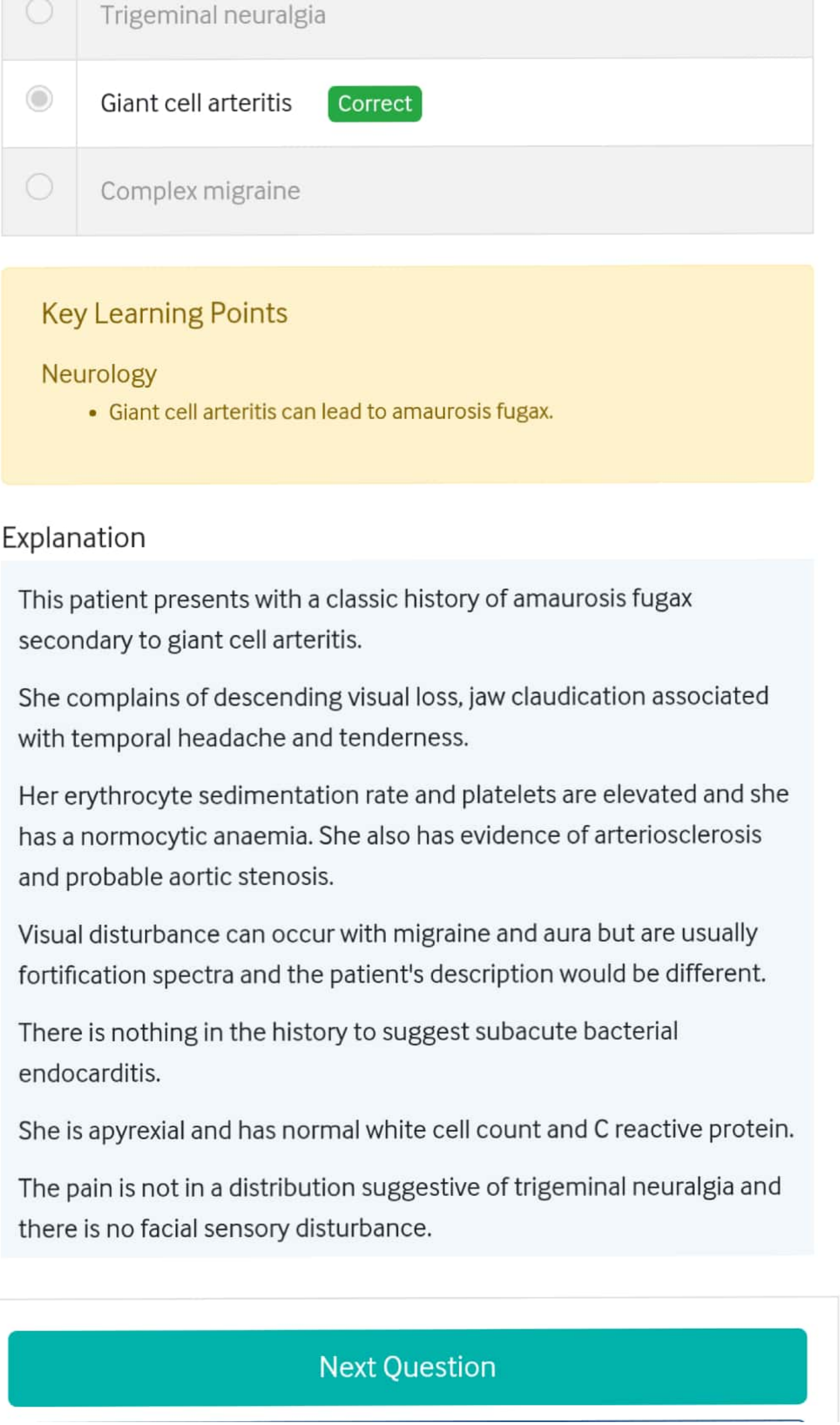
○ Hypertensive retinopathy

○ Trigeminal neuralgia

○ Giant cell arteritis

○ Complex migraine

Submit Answer



○ Trigeminal neuralgia

● Giant cell arteritis **Correct**

○ Complex migraine

Key Learning Points

Neurology

- Giant cell arteritis can lead to amaurosis fugax.

Explanation

This patient presents with a classic history of amaurosis fugax secondary to giant cell arteritis.

She complains of descending visual loss, jaw claudication associated with temporal headache and tenderness.

Her erythrocyte sedimentation rate and platelets are elevated and she has a normocytic anaemia. She also has evidence of arteriosclerosis and probable aortic stenosis.

Visual disturbance can occur with migraine and aura but are usually fortification spectra and the patient's description would be different.

There is nothing in the history to suggest subacute bacterial endocarditis.

She is apyrexial and has normal white cell count and C reactive protein.

The pain is not in a distribution suggestive of trigeminal neuralgia and there is no facial sensory disturbance.

Question 117 of 129

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A 50-year-old woman is referred to you with a year's history of personality change.

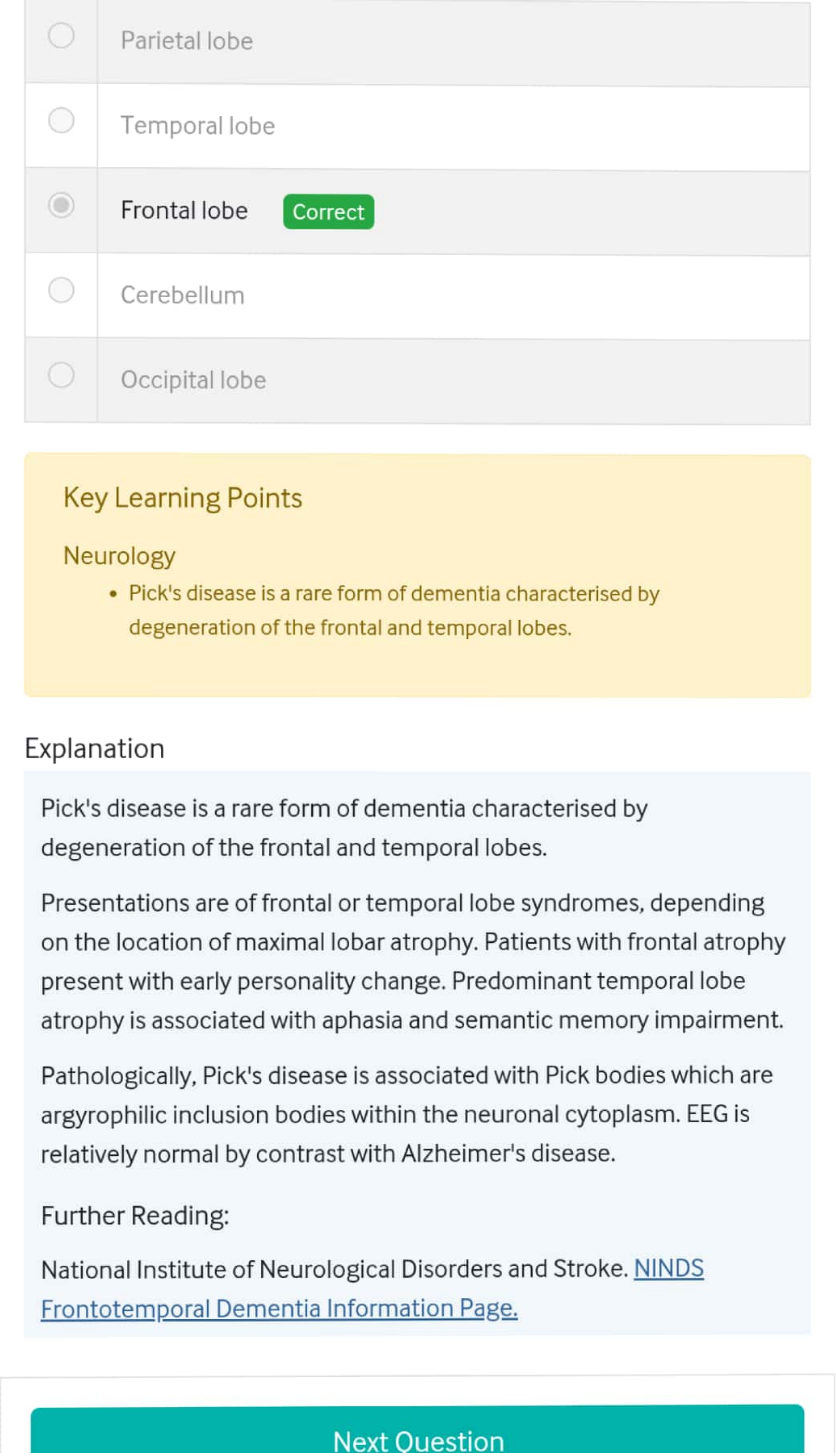
She has become loud, sexually flirtatious and inappropriate in social situations. She has developed increasing difficulties with memory and abstract thinking, but with preservation of arithmetic ability. Speech is relatively preserved and there is no motor impairment.

Which anatomical region of the brain is affected most?

(Please select 1 option)

- ○ Parietal lobe
- ○ Temporal lobe
- ○ Frontal lobe
- ○ Cerebellum
- ○ Occipital lobe

Submit Answer



○ Parietal lobe

○ Temporal lobe

● Frontal lobe

○ Cerebellum

○ Occipital lobe

Key Learning Points

Neurology

- Pick's disease is a rare form of dementia characterised by degeneration of the frontal and temporal lobes.

Explanation

Pick's disease is a rare form of dementia characterised by degeneration of the frontal and temporal lobes.

Presentations are of frontal or temporal lobe syndromes, depending on the location of maximal lobar atrophy. Patients with frontal atrophy present with early personality change. Predominant temporal lobe atrophy is associated with aphasia and semantic memory impairment.

Pathologically, Pick's disease is associated with Pick bodies which are argyrophilic inclusion bodies within the neuronal cytoplasm. EEG is relatively normal by contrast with Alzheimer's disease.

Further Reading:

National Institute of Neurological Disorders and Stroke. NINDS Frontotemporal Dementia Information Page.

A 22-year-old obese woman presented with an eight week history of headaches, pulsatile tinnitus and transient visual loss on standing lasting a few seconds. She had otherwise been well with no history of note.

She took the oral contraceptive pill and had been taking this for the last six months and used salbutamol inhalers on an occasional basis for her asthma which she had from childhood. She also took vitamin A supplements which she bought over the counter for her general health.

On examination, the only abnormality of note was bilateral papilloedema.

MRI brain and MR venogram are normal. Lumbar puncture showed an opening pressure of 38, normal protein, glucose, and cells.

What is the most likely diagnosis?

(Please select 1 option)

○ Sagittal sinus thrombosis secondary to OCP

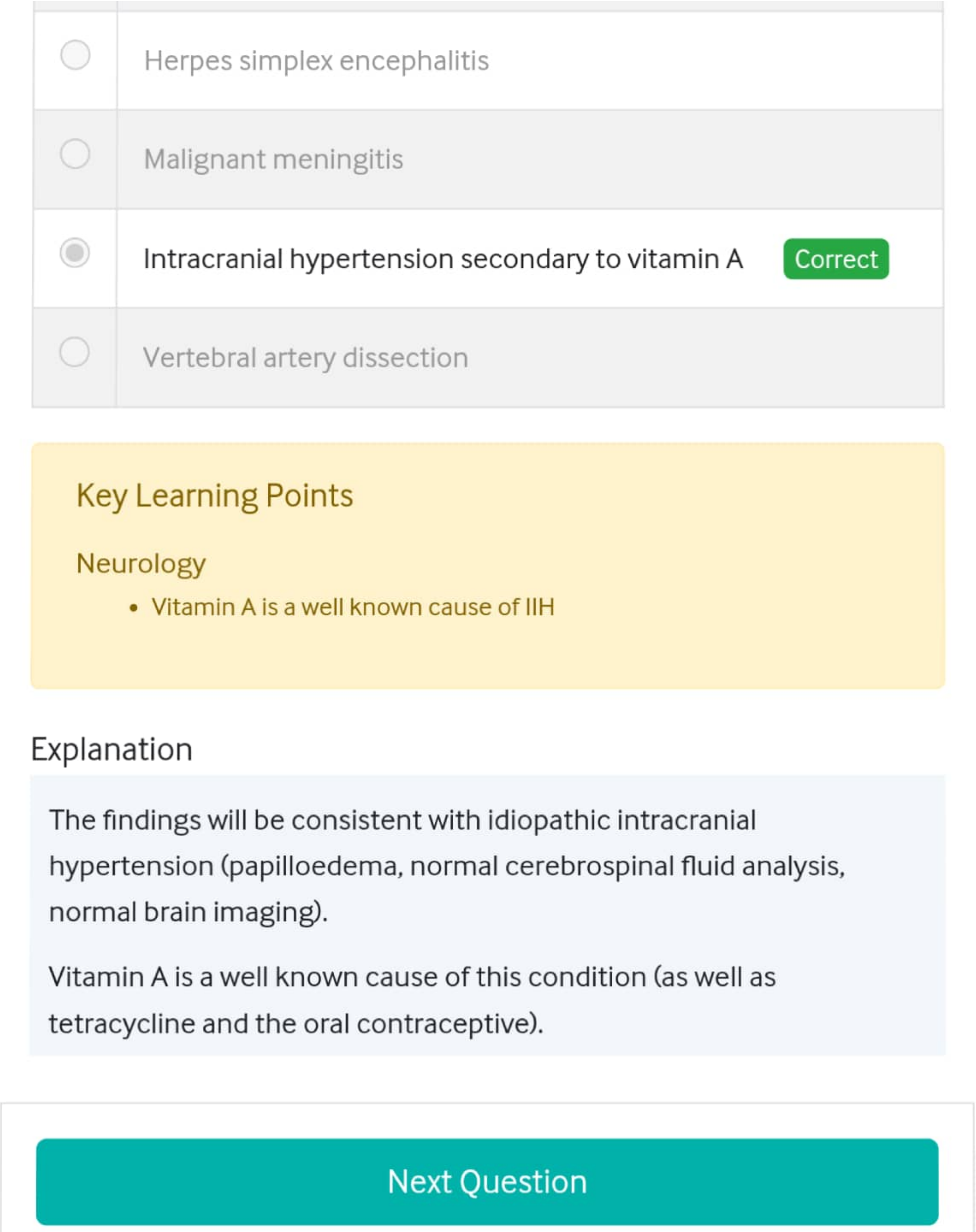
○ Herpes simplex encephalitis

○ Malignant meningitis

○ Intracranial hypertension secondary to vitamin A

○ Vertebral artery dissection

Submit Answer



○ Herpes simplex encephalitis

○ Malignant meningitis

● Intracranial hypertension secondary to vitamin A **Correct**

○ Vertebral artery dissection

Key Learning Points

Neurology

- Vitamin A is a well known cause of IIH

Explanation

The findings will be consistent with idiopathic intracranial hypertension (papilloedema, normal cerebrospinal fluid analysis, normal brain imaging).

Vitamin A is a well known cause of this condition (as well as tetracycline and the oral contraceptive).

Question 119

Question 119 of 129

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A 17-year-old girl was referred for a neurological opinion after suffering several episodes of collapse. Her mother described multiple episodes, lasting from several seconds to one minute, where her daughter would suddenly collapse and "go floppy".

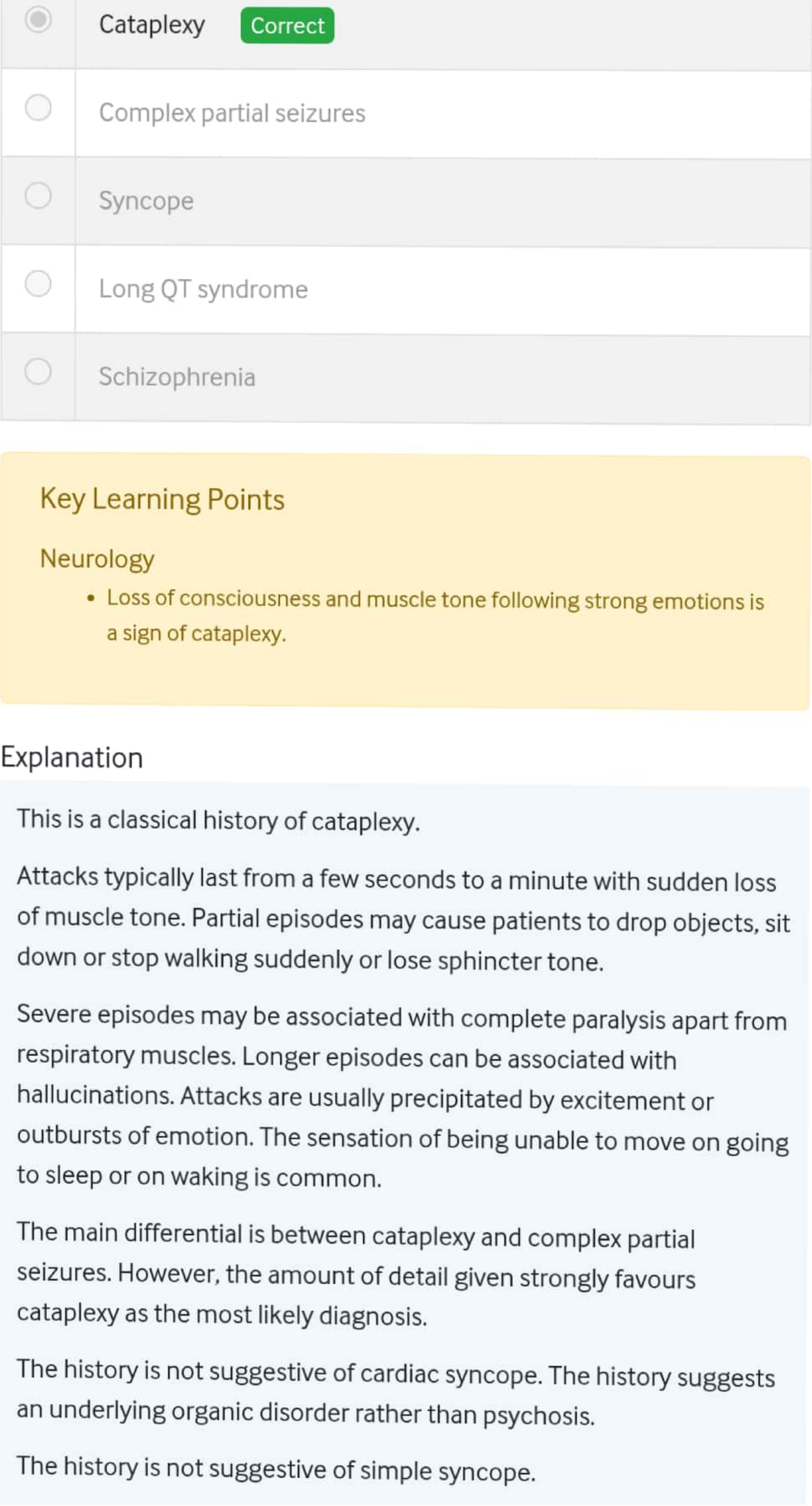
Over the past month she had had one during an argument with her boyfriend; another while watching football on the television and another while she was laughing with her sister. During one episode she experienced visual hallucinations. The patient said that she had never lost consciousness during one of these episodes. She reported that several times she had felt unable to move after waking in the morning.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Cataplexy
- ○ Complex partial seizures
- ○ Syncope
- ○ Long QT syndrome
- ○ Schizophrenia

Submit Answer



- ● Cataplexy **Correct**
- ○ Complex partial seizures
- ○ Syncope
- ○ Long QT syndrome
- ○ Schizophrenia

Key Learning Points

Neurology

- Loss of consciousness and muscle tone following strong emotions is a sign of cataplexy.

Explanation

This is a classical history of cataplexy.

Attacks typically last from a few seconds to a minute with sudden loss of muscle tone. Partial episodes may cause patients to drop objects, sit down or stop walking suddenly or lose sphincter tone.

Severe episodes may be associated with complete paralysis apart from respiratory muscles. Longer episodes can be associated with hallucinations. Attacks are usually precipitated by excitement or outbursts of emotion. The sensation of being unable to move on going to sleep or on waking is common.

The main differential is between cataplexy and complex partial seizures. However, the amount of detail given strongly favours cataplexy as the most likely diagnosis.

The history is not suggestive of cardiac syncope. The history suggests an underlying organic disorder rather than psychosis.

The history is not suggestive of simple syncope.

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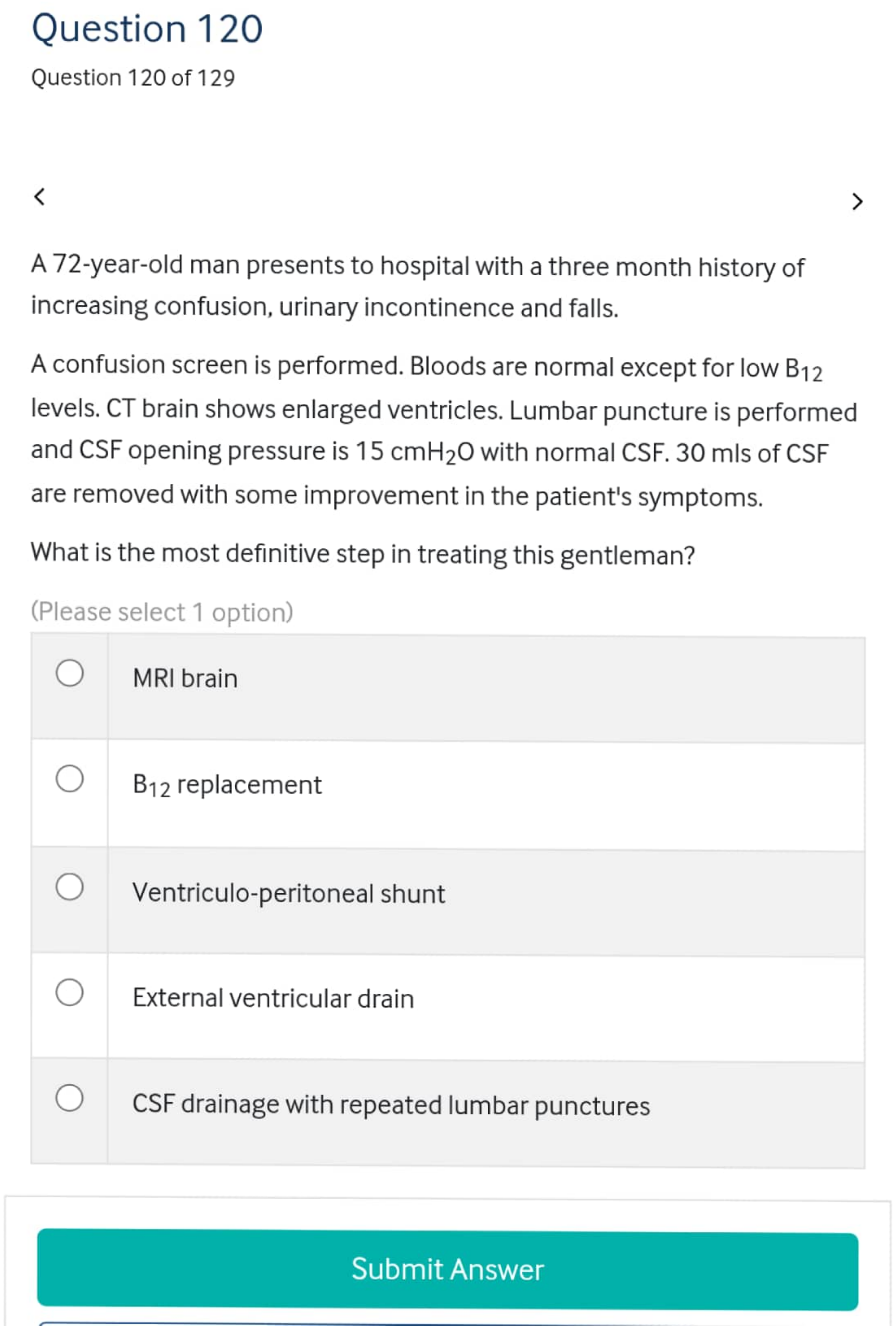


Question 120

Question 120 of 129

A 72-year-old man presents to hospital with a three month history of increasing confusion, urinary incontinence and falls.

A confusion screen is performed. Bloods are normal except for low B_{12} levels. CT brain shows enlarged ventricles. Lumbar puncture is performed and CSF opening pressure is 15 cmH_2O with normal CSF. 30 mls of CSF are removed with some improvement in the patient's symptoms.

What is the most definitive step in treating this gentleman?

(Please select 1 option)

○ MRI brain

○ B_{12} replacement

○ Ventriculo-peritoneal shunt

○ External ventricular drain

○ CSF drainage with repeated lumbar punctures

CamScanner الممسوحة ضوئيا بـ

● Ventriculo-peritoneal shunt **Correct**

○ External ventricular drain

○ CSF drainage with repeated lumbar punctures

Key Learning Points

Neurology

- Normal pressure hydrocephalus characterised by a triad of urinary incontinence, gait disturbance and dementia.

Explanation

This man has normal pressure hydrocephalus characterised by triad of urinary incontinence, gait disturbance and dementia.

His cerebrospinal fluid (CSF) opening pressure is high normal and he has symptomatic improvement with removal of CSF. He should be referred for a ventriculo-peritoneal shunt.

His B_{12} is low and should be replaced but he needs management of the normal pressure hydrocephalus.

CSF drainage with repeated lumbar punctures is an option but can be difficult for patients to tolerate.

External ventricular drain is a temporary method for relieving CSF pressure by draining CSF.

MRI is unlikely to add any further information and will not improve his symptoms.

Next Question

A 30-year-old man presented to the Emergency department with sudden onset weakness affecting the left face, arm and leg. He also complained of left hemi-anaesthesia and restriction of visual fields. He had had generalised headaches in the preceding two weeks but had otherwise been well in himself.

His past medical history included recurrent epistaxis and one recent episode of haemoptysis for which he was awaiting a respiratory opinion. He was a smoker of 20 cigarettes per day and drank 15 units of alcohol per week. He had one sister who had recently been admitted with gastrointestinal bleeding, the cause of which was unclear.

On examination there were punctiform lesions on oral inspection. His blood pressure was 136/78 mmHg, pulse was 65/min and regular and temperature was 36.2°C. Auscultation of the heart and chest did not reveal any abnormalities. Cranial nerve examination revealed a left homonymous hemianopia and left facial weakness with left facial anaesthesia. Peripheral nervous system examination revealed evidence of left sided pyramidal weakness associated with hyperreflexia and hypertonia. Lower limb examination revealed left sided pyramidal weakness with brisk left knee jerk and left extensor plantar. There was left hemisensory loss.

An urgent CT scan brain revealed a right fronto-parietal haemorrhage. Further investigations are outlined below:

Haemoglobin	103 g/L	(130-180)
Mean cell volume	70 fL	(80-96)
White cell count	12.3 ×10^9/L	(4-11)
Platelets	345 ×10^9/L	(150-400)
ESR (Westergren)	10 mm/1st hour	(0-15)
Prothrombin time	14.5 secs	(12-15.5)
APTT	37 secs	(30-46)
Bleeding time	7 mins	(2-8)
Serum sodium	139 mmol/L	(137-144)
Serum potassium	4.1 mmol/L	(3.5-4.9)
Serum urea	3.9 mmol/L	(2.5-7.5)
Serum creatinine	102 µmol/L	(60-110)
Serum cytoplasmic-ANCA	Negative	-
Urinalysis	Blood negative	
	White cells negative	
Gram stain	No organisms seen	
Culture	No growth after 48 hours	

A chest x ray showed patchy shadows within the left lower lobe.

What is the most likely underlying diagnosis?

(Please select 1 option)

○ Klippel-Trenaunay-Weber syndrome

○ von Willebrand's disease

○ Goodpasture's disease

○ Granulomatosis with polyangiitis

○ Hereditary haemorrhagic telangiectasia

Submit Answer

CamScanner الممسوحة ضوئيا بـ

○ Granulomatosis with polyangiitis

● Hereditary haemorrhagic telangiectasia **Correct**

Explanation

This patient presents with left-sided fronto-parietal haemorrhagic stroke as a result of a probable underlying arteriovenous malformation.

He has a history of recent headaches, recurrent epistaxis, and an episode of haemoptysis with probable pulmonary haemorrhages on his chest XR. These findings are highly suggestive of hereditary haemorrhagic telangiectasia (HHT).

Other clues are punctiform oral lesions, a sister with a history of gastrointestinal bleeding in absence of abnormal clotting or thrombocytopenia.

There are two genetic types associated with mutations involving the endoglin gene on chromosome 9q and activin receptor-like kinase 1 located on 12q. HHT typically presents with epistaxis, and arteriovenous malformations of the brain and lung.

Goodpasture's disease is a pulmonary-renal autoimmune syndrome characterised by deposition of antibodies against the glomerular basement membrane.

Klippel-Trenaunay-Weber syndrome is a vascular malformation of the spinal cord associated with a cutaneous naevus and can present with spinal segmental or tract lesions.

von Willebrand's disease is associated with mucosal bleeding and abnormal bleeding time.

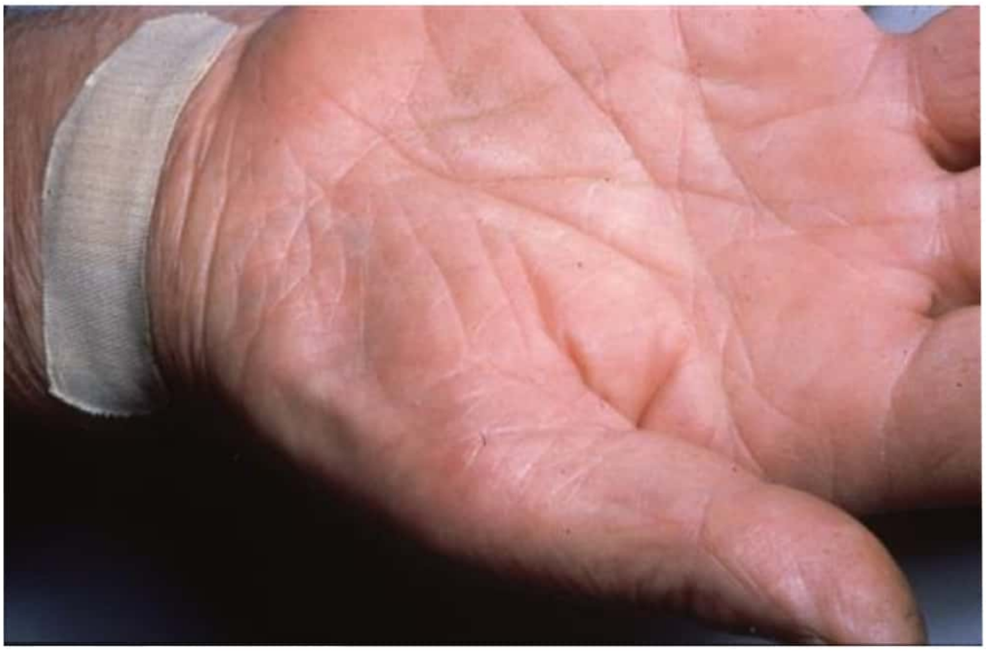
Granulomatosis with polyangiitis is a vasculitis presenting with upper and lower respiratory tract granulomas and glomerulonephritis; cANCA is positive in 70-90% of cases.

CamScanner الممسوحة ضوئيا بـ

A 60-year-old man presents to clinic complaining of a tingling sensation in his right hand extending up the forearm and some weakness of his right thumb. He had had the problem for several months.

He had a longstanding history of hypothyroidism and rheumatoid arthritis and took thyroxine 75 mg/day and azathioprine.

On examination, there was reduced sensation to light touch over the lateral three and a half digits on the palmar aspect of his right hand. Sensation over the thenar and hypothenar eminences was normal, as was sensation in the medial one and a half fingers. There was also loss of power of right thumb flexion and opposition but no demonstrable deficit in the other fingers.



Which of the following muscles are likely to be affected in this patient?

(Please select 1 option)

- ○ Palmar interossei
- ○ Abductor digiti minimi
- ○ Dorsal interossei
- ○ Fourth lumbrical
- ○ Flexor pollicis brevis

Submit Answer

CamScanner الممسوحة ضوئيا بـ

○ Flexor pollicis brevis **Correct**

Key Learning Points

Neurology

- Median nerve supplies the LOAF muscles: Lateral 2 lumbricals, Opponens pollicis, ABductor pollicis brevis, Flexor pollicis brevis

Explanation

The slide shows wasting of the thenar eminence.

The diagnosis is carpal tunnel syndrome, which arises due to pressure on the median nerve in the carpal tunnel. The precise cause of the syndrome is unknown, though it is likely that thickening of the synovial sheaths of the flexor tendons and arthritic changes in the carpal bones contribute.

The ulnar nerve carries sensory fibres from the palmar aspect of the medial one and a half fingers. The median nerve innervates the palmar aspect of the lateral three and a half fingers. Sensation to the thenar eminence is supplied via the palmar cutaneous branch of the median nerve, which passes superficially to the flexor retinaculum and is therefore unaffected by carpal tunnel syndrome.

The median nerve also supplies most of the muscles of the thenar eminence, namely the:

- Lateral two lumbricals
- Opponens pollicis
- Abductor pollicis brevis, and
- Flexor pollicis brevis

These can be remembered with the mnemonic LOAF.

The remaining small muscles of the hand are supplied by the ulnar nerve.

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A 19-year-old farm worker was admitted with a two day history of progressive weakness and tingling of the extremities affecting all limbs.

He had been well until a week ago when he had an episode of running nose, cough and a sore throat which resolved after 48 hours without therapy.

On examination, pulse rate was 106 beats/min, BP 130/75 mmHg and respiratory rate 16/min. Cranial nerve examination was unremarkable. He had decreased muscle strength in all extremities, lower more than upper, and absent deep tendon reflexes in the legs.

Investigations showed

Hb	146 g/L	(130-180)
WCC	9.3×10^9/L	(4-11)
Platelets	400×10^9/L	(150-400)
ESR	2 mm/hr	(0-15)
Plasma sodium	135 mmol/L	(137-144)
Plasma potassium	3.8 mmol/L	(3.5-4.9)
Plasma urea	6.0 mmol/L	(2.5-7.5)
Plasma creatinine	100 µmol/L	(60-110)
Plasma glucose	5.0 mmol/L	(3.0-6.0)
CSF opening pressure	15 cm H_2O	(50-180)
CSF cell count	<2 cells/ml	($\leq$ 5 mL^{-1})
CSF protein	1.5 g/L	(0.15-0.45)
CSF glucose	6.1 mmol/L	(3.0-6.0)

What is the most important parameter that needs to be monitored in this patient?

(Please select 1 option)

○ Peak expiratory flow rate

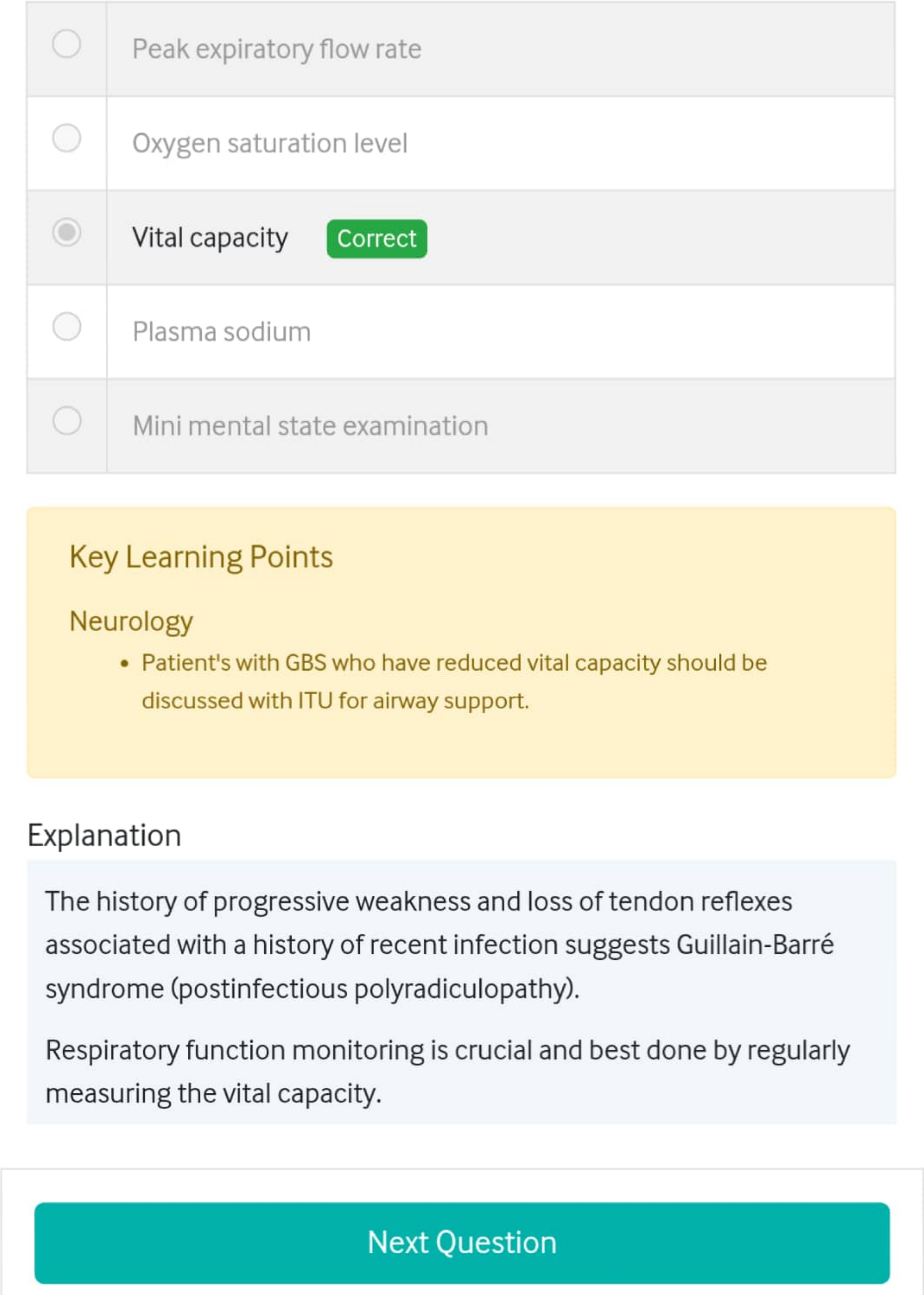
○ Oxygen saturation level

○ Vital capacity

○ Plasma sodium

○ Mini mental state examination

CamScanner الممسوحة ضوئيا بـ



○ Peak expiratory flow rate

○ Oxygen saturation level

● Vital capacity

○ Plasma sodium

○ Mini mental state examination

Key Learning Points

Neurology

- Patient's with GBS who have reduced vital capacity should be discussed with ITU for airway support.

Explanation

The history of progressive weakness and loss of tendon reflexes associated with a history of recent infection suggests Guillain-Barré syndrome (postinfectious polyradiculopathy).

Respiratory function monitoring is crucial and best done by regularly measuring the vital capacity.

CamScanner الممسوحة ضوئيا بـ

A 47-year-old woman was seen in general medical clinic complaining of a three month history of progressive diffuse myalgia and weakness.

The weakness appeared to affect her shoulder muscles and thighs, and she had noticed she was experiencing great difficulty in walking up and down stairs. She had not noticed any weakness. She had a past medical history of hypertension and hyperlipidaemia and took regular atenolol and simvastatin.

Examination of the cranial nerves revealed no abnormalities and there was no detectable neck weakness.

On upper limb examination there was general myalgia, but no evidence of wasting. Tone, reflexes and sensation were all normal, but there was proximal weakness of 3/5 with preserved distal power. There was a similar pattern of weakness in the lower limb with preserved tone, reflexes and sensation.

Investigations showed:

Haemoglobin	124 g/L	(130-180)
White cell count	6.0 $\times 10^9$/L	(4-11)
Platelets	267 $\times 10^9$/L	(150-400)
ESR (Westergren)	15 mm/1st hour	(0-15)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	4.8 mmol/L	(3.5-4.9)
Serum urea	6.2 mmol/L	(2.5-7.5)
Serum creatinine	120 µmol/L	(60-110)
Plasma lactate	1.2 mmol/L	(0.6-1.8)
Serum creatine kinase	599 U/L	(24-170)
Fasting plasma glucose	4.6 mmol/L	(3-6)
Serum cholesterol	3.6 mmol/L	(<5.2)
Plasma TSH	4.1 mU/L	(0.5-5.0)
Plasma T4	63 nmol/L	(58-174)
Plasma T3	2.6 nmol/L	(1.07-3.18)

Urinalysis was normal.

What is the likely diagnosis?

(Please select 1 option)

○ Statin-induced myopathy

○ McArdle's disease

○ Neuroleptic malignant syndrome

○ Mitochondrial myopathy

○ Inclusion body myositis

● Statin-induced myopathy **Correct**

○ McArdle's disease

○ Neuroleptic malignant syndrome

○ Mitochondrial myopathy

○ Inclusion body myositis

Key Learning Points

Emergency Medicine, Neurology, Therapeutics

- Myalgia, myositis and myopathy are all recognised effects of HMG-CoA reductase inhibitors (statins). There is an increased incidence when statins are co-administered with a fibrate or when given to patients on immunosuppressants.

Explanation

Myalgia, myositis and myopathy are all recognised effects of HMG-CoA reductase inhibitors (statins). There is an increased incidence when statins are co-administered with a fibrate or when given to patients on immunosuppressants. Unless therapy is withdrawn, rhabdomyolysis may ensue often associated with acute renal failure secondary to myoglobinuria.

Inclusion body myositis is an inflammatory myopathy producing distal and asymmetric weakness.

McArdle's disease tends to present in children with painful muscle cramps and myoglobinuria after intense exercise. It is an autosomal recessive condition resulting myophosphorylase deficiency which leads to an inability to utilise glucose.

There are no extra neurological features to suggest a mitochondrial disorder and the plasma lactate is normal.

Neuroleptic malignant syndrome is caused by anti-psychotic medication.

Next Question

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A 58-year-old gentleman comes to see you. You had previously referred him to secondary care when he presented with a progressively worsening tremor. Parkinson's disease was suspected and investigation in secondary care confirmed the diagnosis.

Over the last 18 months his condition has worsened and he has started to develop significant functional disability. He has spoken to his Parkinson's disease specialist nurse who has suggested he might need to start drug treatment. She has arranged for him to be seen by his consultant in outpatients to discuss this.

He has been reading a lot of information online but has found it all very confusing. He was not sure which type of drug the consultant might advise and wanted to discuss the likely options.

Which of the following classes of drug would be an appropriate option for initial pharmacotherapy in this patient?

(Please select 1 option)

○	Levodopa
○	Anticholinergic
○	Amantadine
○	Catecholamine-o-methyl transferase inhibitor
○	Beta blocker

Submit Answer

(Please select 1 option)

●	Levodopa **Correct**
○	Anticholinergic
○	Amantadine
○	Catecholamine-o-methyl transferase inhibitor
○	Beta blocker

Key Learning Points

Neurology

- Fist choice options for initial pharmacotherapy in people with early Parkinson’s disease are levodopa, dopamine agonists and MAO-B inhibitors.

Explanation

The pharmacological management options in Parkinson's disease (PD) can be confusing. GPs may be asked to prescribe these drugs although usually they would be initiated in secondary care. It is important to have an understanding of which drugs are used as patients may seek advice about treatment options or may present to you with a drug side effect, for example.

The NICE guidance emphasizes that it is not possible to advise a single first line pharmacological option in someone with early PD and the choice of drug needs to individualized taking into account the clinical picture, the lifestyle of the patient, and patient preference.

There is a lack of evidence for the effectiveness of amantadine, anticholinergics, and beta blockers as first line options in the management of early PD. COMT inhibitors may have a role as an adjuvant treatment in later PD but not in initial pharmacotherapy.

Levodopa, dopamine agonists, and MAO-B inhibitors are the first choice options as there is evidence behind their use in providing symptoms control.

A 62-year-old woman is referred with a three month history of increasing weakness affecting the left more than the right arm. She also complains of tingling and numbness affecting the left thumb which tends to be worse at nights.

She has dropped cups of tea twice in the last month but attributes this to her own clumsiness. She denies any visual, speech, bowel or bladder symptoms. Occasionally she gets some neck discomfort on turning her head but denies any recent or past history of neck trauma. Past medical history includes a left mastectomy 10 years ago for breast cancer. Her sister died of a brain tumour.

On examination, her cranial nerves are intact. No increase in tone is detectable in all limbs. She has mild weakness when flexing the supinated forearm against resistance, diminished biceps and brachioradialis reflexes and increased triceps reflexes bilaterally. The rest of her muscle power and reflexes are normal. Plantar responses are flexor bilaterally. Sensory examination is unremarkable to pinprick and joint position sense.

What is the most likely diagnosis in this woman?

(Please select 1 option)

- ○ Multiple sclerosis
- ○ Hereditary spastic paraparesis
- ○ Ulnar nerve entrapment
- ○ Amyotrophic lateral sclerosis
- ○ Cervical myelopathy

Submit Answer

○ Amyotrophic lateral sclerosis

● Cervical myelopathy **Correct**

Key Learning Points

Neurology

- Nerve root lesions will present with sign and symptoms affecting a specific dermatome and myotome.

Explanation

The symptoms in this patient best fit a spondylotic problem affecting the C5/6 level, thus the C6 cervical root.

Symptoms and signs of a C6 root lesion include

- Paraesthesias in the thumb or lateral distal forearm
- Weakness of brachioradialis, biceps, or triceps and
- Diminished biceps and brachioradialis reflexes in conjunction with an increased triceps reflex.

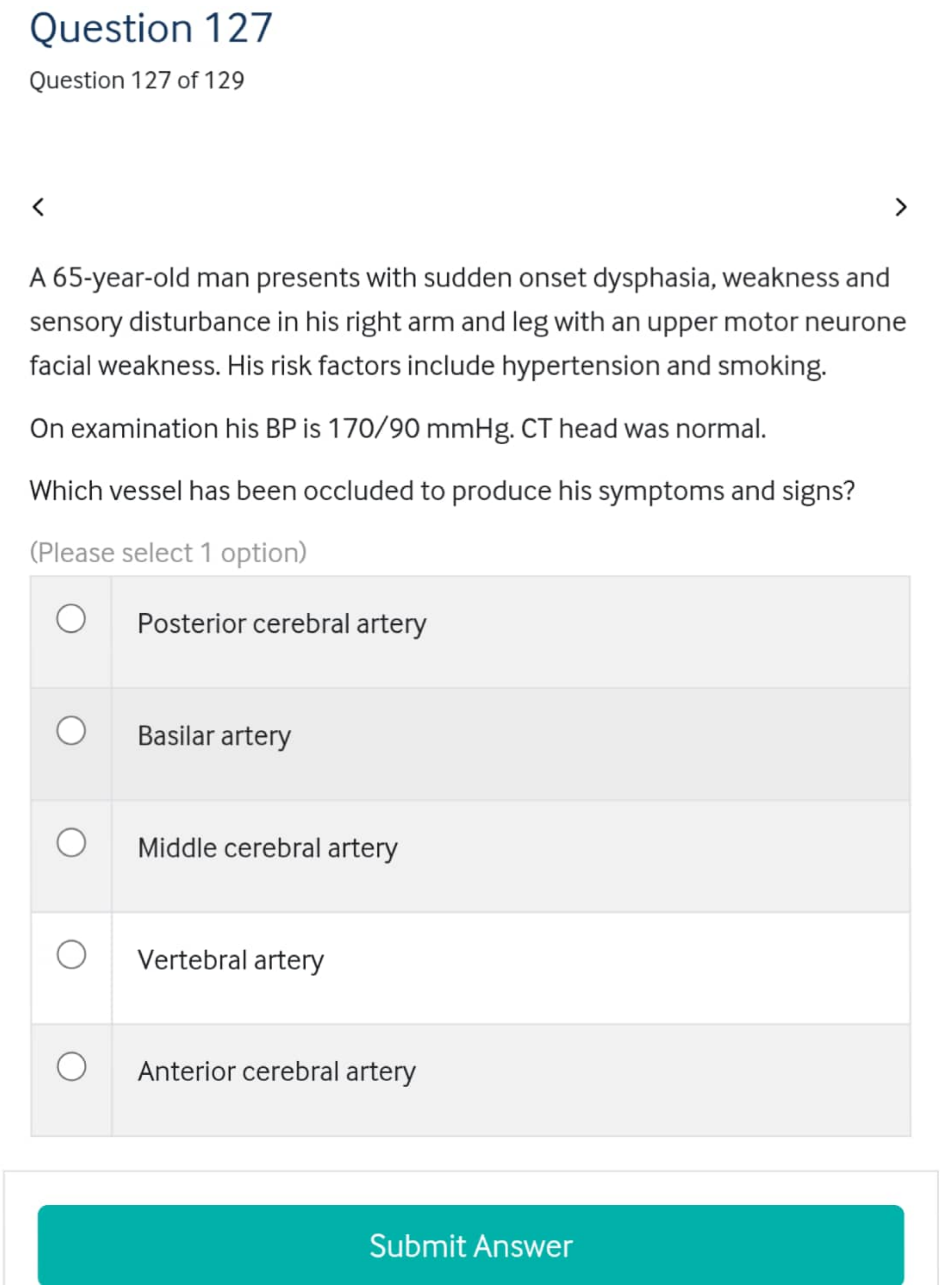
Patients do not always present with all of these features but the key finding here is the inverted upper limb reflexes (brisk triceps and diminished biceps and brachioradialis).

The presence of sensory symptoms make amyotrophic lateral sclerosis unlikely.

Ulnar nerve entrapment does not cause reflex changes.

Hereditary spastic paraparesis usually begins with in the lower limbs and the patient is a bit old for it to start manifesting only now. Multiple sclerosis is also less common in this age group.

Next Question



Question 127

Question 127 of 129

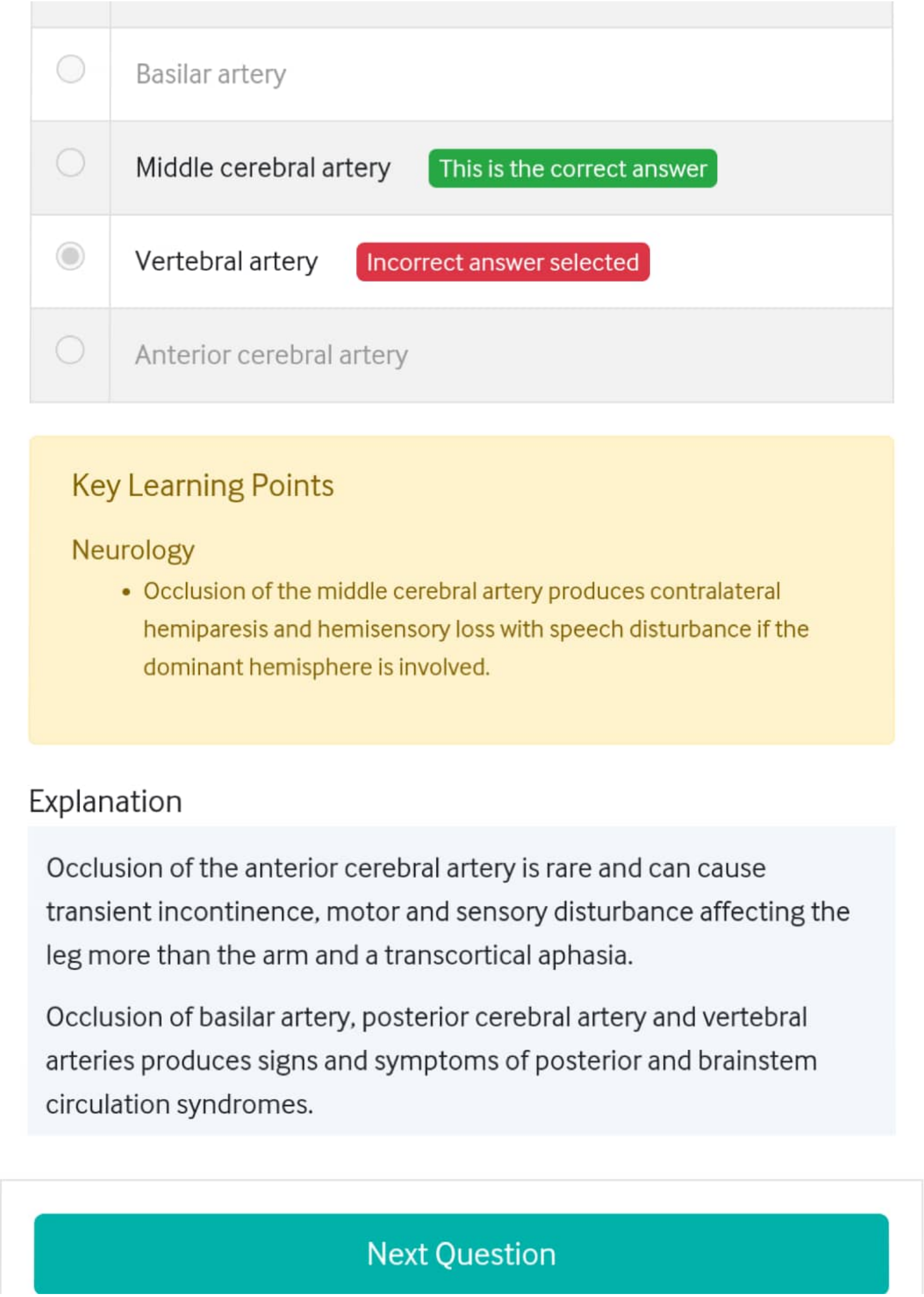
A 65-year-old man presents with sudden onset dysphasia, weakness and sensory disturbance in his right arm and leg with an upper motor neurone facial weakness. His risk factors include hypertension and smoking.

On examination his BP is 170/90 mmHg. CT head was normal.

Which vessel has been occluded to produce his symptoms and signs?

(Please select 1 option)

- ○ Posterior cerebral artery
- ○ Basilar artery
- ○ Middle cerebral artery
- ○ Vertebral artery
- ○ Anterior cerebral artery



○ Basilar artery

○ Middle cerebral artery This is the correct answer

● Vertebral artery Incorrect answer selected

○ Anterior cerebral artery

Key Learning Points

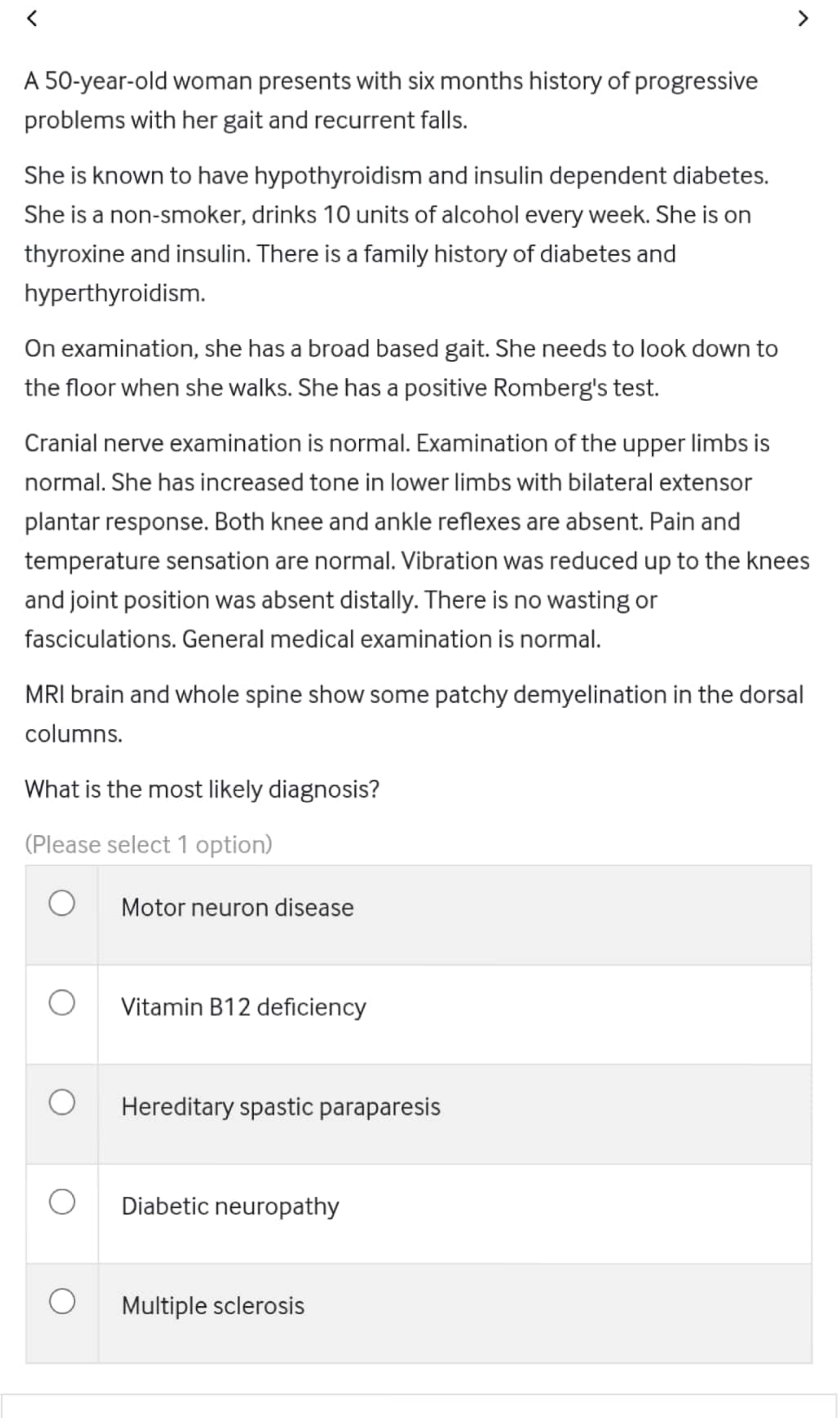
Neurology

- Occlusion of the middle cerebral artery produces contralateral hemiparesis and hemisensory loss with speech disturbance if the dominant hemisphere is involved.

Explanation

Occlusion of the anterior cerebral artery is rare and can cause transient incontinence, motor and sensory disturbance affecting the leg more than the arm and a transcortical aphasia.

Occlusion of basilar artery, posterior cerebral artery and vertebral arteries produces signs and symptoms of posterior and brainstem circulation syndromes.



A 50-year-old woman presents with six months history of progressive problems with her gait and recurrent falls.

She is known to have hypothyroidism and insulin dependent diabetes. She is a non-smoker, drinks 10 units of alcohol every week. She is on thyroxine and insulin. There is a family history of diabetes and hyperthyroidism.

On examination, she has a broad based gait. She needs to look down to the floor when she walks. She has a positive Romberg's test.

Cranial nerve examination is normal. Examination of the upper limbs is normal. She has increased tone in lower limbs with bilateral extensor plantar response. Both knee and ankle reflexes are absent. Pain and temperature sensation are normal. Vibration was reduced up to the knees and joint position was absent distally. There is no wasting or fasciculations. General medical examination is normal.

MRI brain and whole spine show some patchy demyelination in the dorsal columns.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Motor neuron disease
- ○ Vitamin B12 deficiency
- ○ Hereditary spastic paraparesis
- ○ Diabetic neuropathy
- ○ Multiple sclerosis

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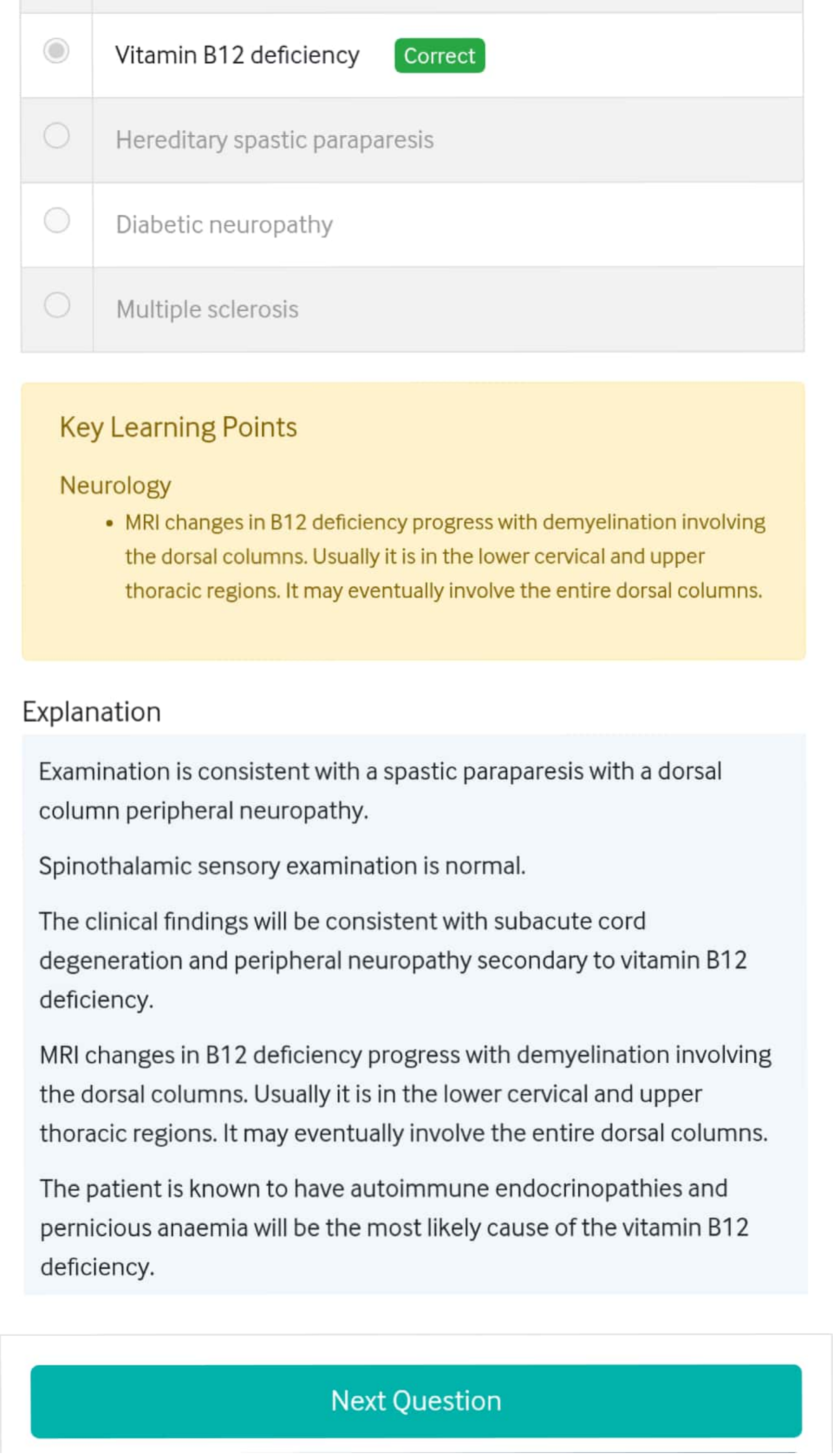


● Vitamin B12 deficiency Correct

○ Hereditary spastic paraparesis

○ Diabetic neuropathy

○ Multiple sclerosis

Key Learning Points

Neurology

- MRI changes in B12 deficiency progress with demyelination involving the dorsal columns. Usually it is in the lower cervical and upper thoracic regions. It may eventually involve the entire dorsal columns.

Explanation

Examination is consistent with a spastic paraparesis with a dorsal column peripheral neuropathy.

Spinothalamic sensory examination is normal.

The clinical findings will be consistent with subacute cord degeneration and peripheral neuropathy secondary to vitamin B12 deficiency.

MRI changes in B12 deficiency progress with demyelination involving the dorsal columns. Usually it is in the lower cervical and upper thoracic regions. It may eventually involve the entire dorsal columns.

The patient is known to have autoimmune endocrinopathies and pernicious anaemia will be the most likely cause of the vitamin B12 deficiency.

A 20-year-old man was seen in neurology clinic having developed stiffness and weakness of both legs over several months. He had not noticed any problems with his bowel or bladder and denied any sensory loss in the legs or back trauma. He had also noticed mild weakness of the left hand with reduced dexterity.

He had a past medical history of delayed milestones and generalised epilepsy and was taking regular phenytoin. He was a non-smoker and did not drink any alcohol. There was a family history of epilepsy.

On examination he had mild learning difficulties. His vital signs were all normal. Examination of cranial nerves revealed bilateral retinal hamartomas however pupillary responses and ocular movements were intact. The rest of the cranial nerves were normal.

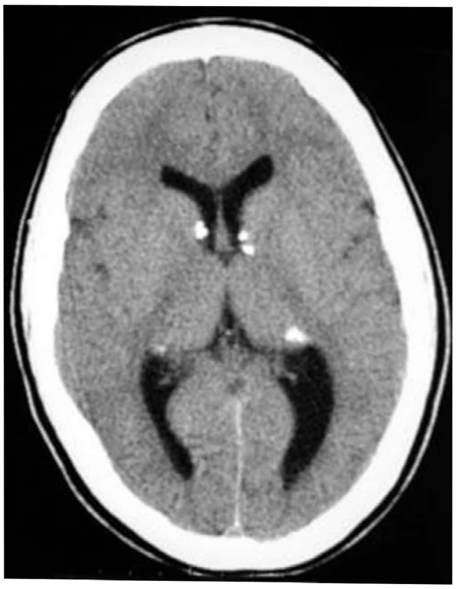
Upper limb examination revealed mildly increased tone on the left with brisk left sided reflexes and weakness of left hand grip. Power on the right was normal. Examination of the lower limb revealed bilaterally increased tone with mild pyramidal weakness and hyper-reflexia. Plantar responses were both extensor. There were no abnormalities detected on examining sensation or cerebellar function.

There were several raised lesions affecting the face and some brown coarse patches over the trunk.

Investigations revealed:

Haemoglobin	146 g/L	(130-180)
White cell count	6.1 ×10^9/L	(4-11)
Platelets	187 ×10^9/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	6.7 mmol/L	(2.5-7.5)
Serum creatinine	120 µmol/L	(60-110)
Serum corrected calcium	2.6 mmol/L	(2.2-2.6)
Serum phosphate	0.8 mmol/L	(0.8-1.4)
Serum AST	27 U/L	(1-31)
Serum alanine aminotransferase	32 U/L	(5-35)
Serum alkaline phosphatase	105 U/L	(45-105)

A CT brain scan was performed. An image from this study, taken before the administration of intravenous contrast, is shown below:



Given the clinical account and investigations performed so far, what is the likely diagnosis?

(Please select 1 option)

○ Neurofibromatosis type I

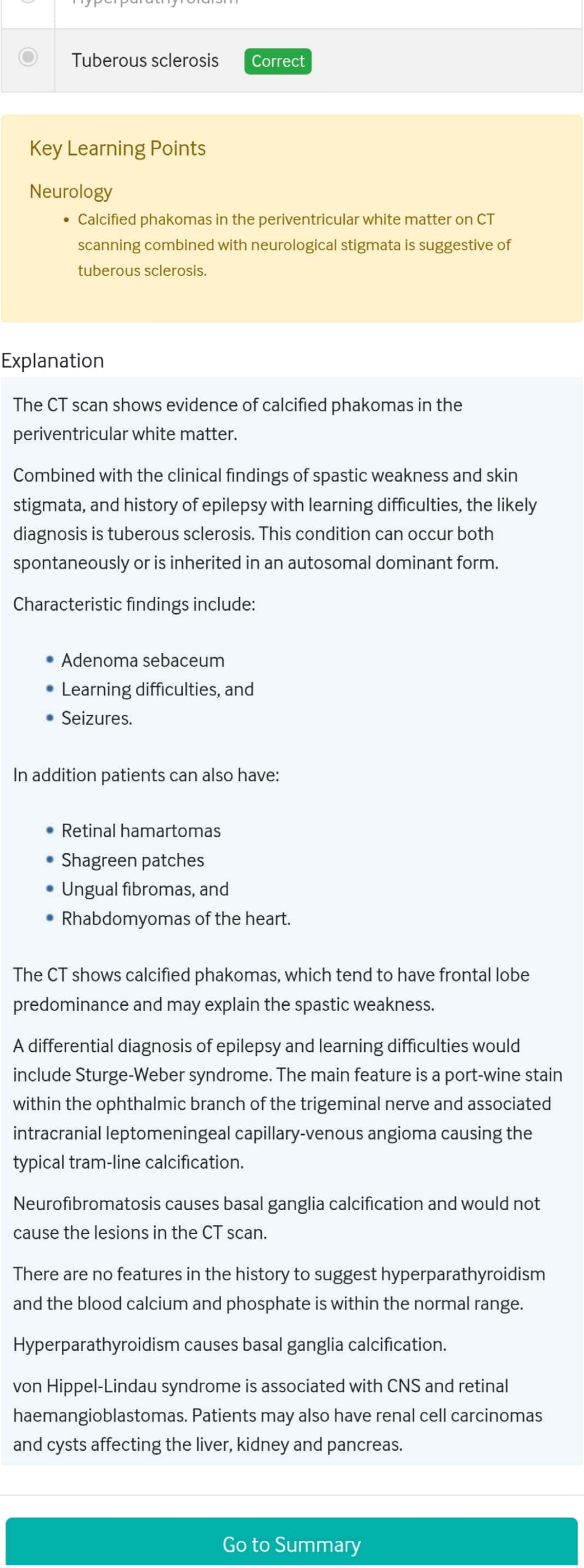
○ Sturge-Weber syndrome

○ von Hippel-Lindau syndrome

○ Hyperparathyroidism

○ Tuberous sclerosis

Submit Answer



○ Hyperparathyroidism

● Tuberous sclerosis **Correct**

Key Learning Points

Neurology

- Calcified phakomas in the periventricular white matter on CT scanning combined with neurological stigmata is suggestive of tuberous sclerosis.

Explanation

The CT scan shows evidence of calcified phakomas in the periventricular white matter.

Combined with the clinical findings of spastic weakness and skin stigmata, and history of epilepsy with learning difficulties, the likely diagnosis is tuberous sclerosis. This condition can occur both spontaneously or is inherited in an autosomal dominant form.

Characteristic findings include:

- Adenoma sebaceum
- Learning difficulties, and
- Seizures.

In addition patients can also have:

- Retinal hamartomas
- Shagreen patches
- Ungual fibromas, and
- Rhabdomyomas of the heart.

The CT shows calcified phakomas, which tend to have frontal lobe predominance and may explain the spastic weakness.

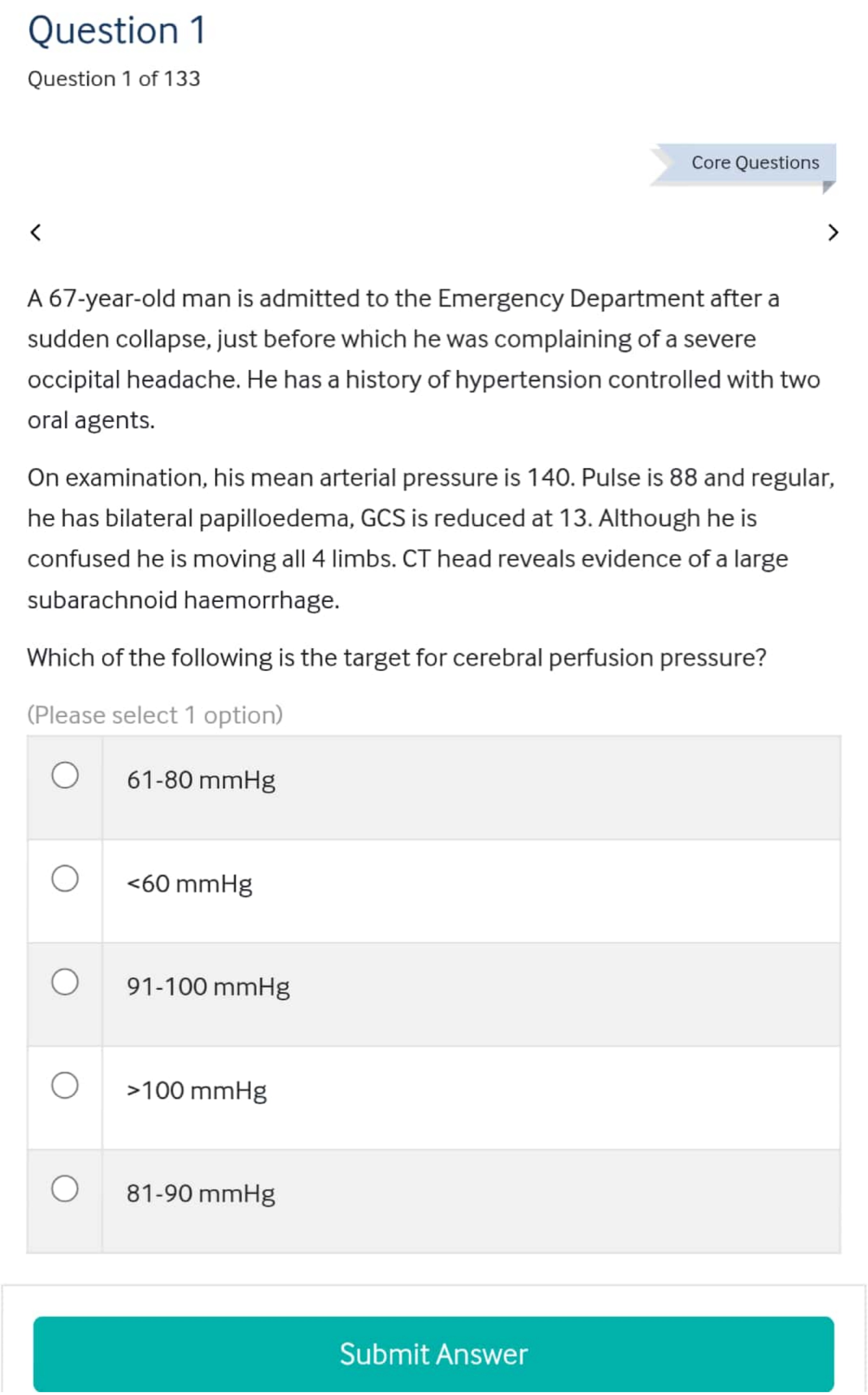
A differential diagnosis of epilepsy and learning difficulties would include Sturge-Weber syndrome. The main feature is a port-wine stain within the ophthalmic branch of the trigeminal nerve and associated intracranial leptomeningeal capillary-venous angioma causing the typical tram-line calcification.

Neurofibromatosis causes basal ganglia calcification and would not cause the lesions in the CT scan.

There are no features in the history to suggest hyperparathyroidism and the blood calcium and phosphate is within the normal range.

Hyperparathyroidism causes basal ganglia calcification.

von Hippel-Lindau syndrome is associated with CNS and retinal haemangioblastomas. Patients may also have renal cell carcinomas and cysts affecting the liver, kidney and pancreas.



Question 1

Question 1 of 133

A 67-year-old man is admitted to the Emergency Department after a sudden collapse, just before which he was complaining of a severe occipital headache. He has a history of hypertension controlled with two oral agents.

On examination, his mean arterial pressure is 140. Pulse is 88 and regular, he has bilateral papilloedema, GCS is reduced at 13. Although he is confused he is moving all 4 limbs. CT head reveals evidence of a large subarachnoid haemorrhage.

Which of the following is the target for cerebral perfusion pressure?

(Please select 1 option)

○	61-80 mmHg
○	<60 mmHg
○	91-100 mmHg
○	>100 mmHg
○	81-90 mmHg

Which of the following is the target for cerebral perfusion pressure?

(Please select 1 option)

● 61-80 mmHg **Correct**

○ <60 mmHg

○ 91-100 mmHg

○ >100 mmHg

○ 81-90 mmHg

Key Learning Points

Neurology, Stroke

- A cerebral perfusion pressure of 61-80 mmHg is recommended in SAH to prevent watershed infarcts as well as worsening of the bleed.

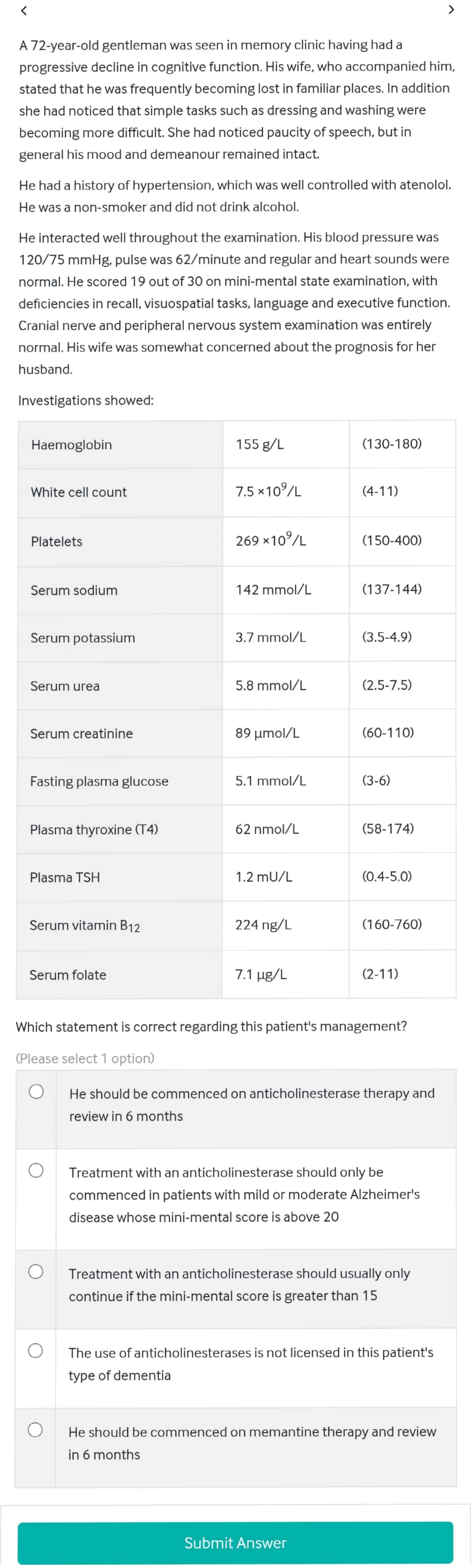
Explanation

The target is 61-80 mmHg. The most relevant guidelines come from the American Heart Association and American Stroke Association, who recommend a cerebral perfusion pressure of 61-80 mmHg. Perfusion pressures below 60 run the risk of inadequate perfusion and watershed infarction, and pressures >80 mmHg run the risk of extending any area of cerebral haemorrhage.

The other options are either incorrect because they run the risk of expanding any area of neurological impairment related to poor blood supply, or extending the cerebral haemorrhage.

Evidence for the level of blood pressure lowering required is actually remarkably weak, with one main trial, (INTERACT2), looking at aggressive BP lowering, (to less than 140mmHg), vs standard management of blood pressure. This study did not show a significant improvement in hard outcomes, although a secondary measure, (Rankin score), was better in patients with BP between 150 and 200 mmHg systolic who were targeted to the aggressive lowering group.

IV agents are preferred for lowering blood pressure in the acute phase as their dose can be easily titrated.



A 72-year-old gentleman was seen in memory clinic having had a progressive decline in cognitive function. His wife, who accompanied him, stated that he was frequently becoming lost in familiar places. In addition she had noticed that simple tasks such as dressing and washing were becoming more difficult. She had noticed paucity of speech, but in general his mood and demeanour remained intact.

He had a history of hypertension, which was well controlled with atenolol. He was a non-smoker and did not drink alcohol.

He interacted well throughout the examination. His blood pressure was 120/75 mmHg, pulse was 62/minute and regular and heart sounds were normal. He scored 19 out of 30 on mini-mental state examination, with deficiencies in recall, visuospatial tasks, language and executive function. Cranial nerve and peripheral nervous system examination was entirely normal. His wife was somewhat concerned about the prognosis for her husband.

Investigations showed:

Haemoglobin	155 g/L	(130-180)
White cell count	7.5 $\times 10^9$/L	(4-11)
Platelets	269 $\times 10^9$/L	(150-400)
Serum sodium	142 mmol/L	(137-144)
Serum potassium	3.7 mmol/L	(3.5-4.9)
Serum urea	5.8 mmol/L	(2.5-7.5)
Serum creatinine	89 µmol/L	(60-110)
Fasting plasma glucose	5.1 mmol/L	(3-6)
Plasma thyroxine (T4)	62 nmol/L	(58-174)
Plasma TSH	1.2 mU/L	(0.4-5.0)
Serum vitamin B_{12}	224 ng/L	(160-760)
Serum folate	7.1 µg/L	(2-11)

Which statement is correct regarding this patient's management?

(Please select 1 option)

○ He should be commenced on anticholinesterase therapy and review in 6 months

○ Treatment with an anticholinesterase should only be commenced in patients with mild or moderate Alzheimer's disease whose mini-mental score is above 20

○ Treatment with an anticholinesterase should usually only continue if the mini-mental score is greater than 15

○ The use of anticholinesterases is not licensed in this patient's type of dementia

○ He should be commenced on memantine therapy and review in 6 months

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Which statement is correct regarding this patient's management?

(Please select 1 option)

● He should be commenced on anticholinesterase therapy and review in 6 months **Correct**

○ Treatment with an anticholinesterase should only be commenced in patients with mild or moderate Alzheimer's disease whose mini-mental score is above 20

○ Treatment with an anticholinesterase should usually only continue if the mini-mental score is greater than 15

○ The use of anticholinesterases is not licensed in this patient's type of dementia

○ He should be commenced on memantine therapy and review in 6 months

Key Learning Points

Neurology

- Memantine is recommended for use in severe Alzhemier's or moderate disease where AChe inhibitors are not tolerated or contraindicated.

Explanation

This patient has features in keeping with Alzheimer's disease (AD).

NICE guidelines on Alzheimer's disease and the pharmacological management of dementia suggest the following:

The three acetylcholinesterase (AChE) inhibitors donepezil, galantamine and rivastigmine are recommended as options for managing mild to moderate Alzheimer's disease under all of the conditions specified.

Memantine is recommended as an option for managing Alzheimer's disease for people with:

- moderate Alzheimer's disease who are intolerant of or have a contraindication to AChE inhibitors, or
- severe Alzheimer's disease.

Repeating the cognitive assessment every 6 months assesses benefit. Up to half the patients given these drugs will show a slower rate of cognitive decline. The drug should be discontinued in those thought not to be responding. In those who are responding, drugs should be stopped once the MMSE score is less than 10.

An 81-year-old lady presents with lethargy and poor mobility. Her husband called the ambulance after he had problems waking her up in the morning. She would only open her eyes to his voice for a few seconds before going back to sleep.

She normally walks with a Zimmer frame and although normally steady on her feet had fallen one week prior to admission.

Her past medical history included hypertension and diabetes.

On examination her Glasgow coma scale was reduced at 13/15. Planters were upgoing bilaterally. An urgent CT head is organised which shows evidence of bilateral subdural haematomas.

Which vessel is involved?

(Please select 1 option)

○ Anterior communicating artery

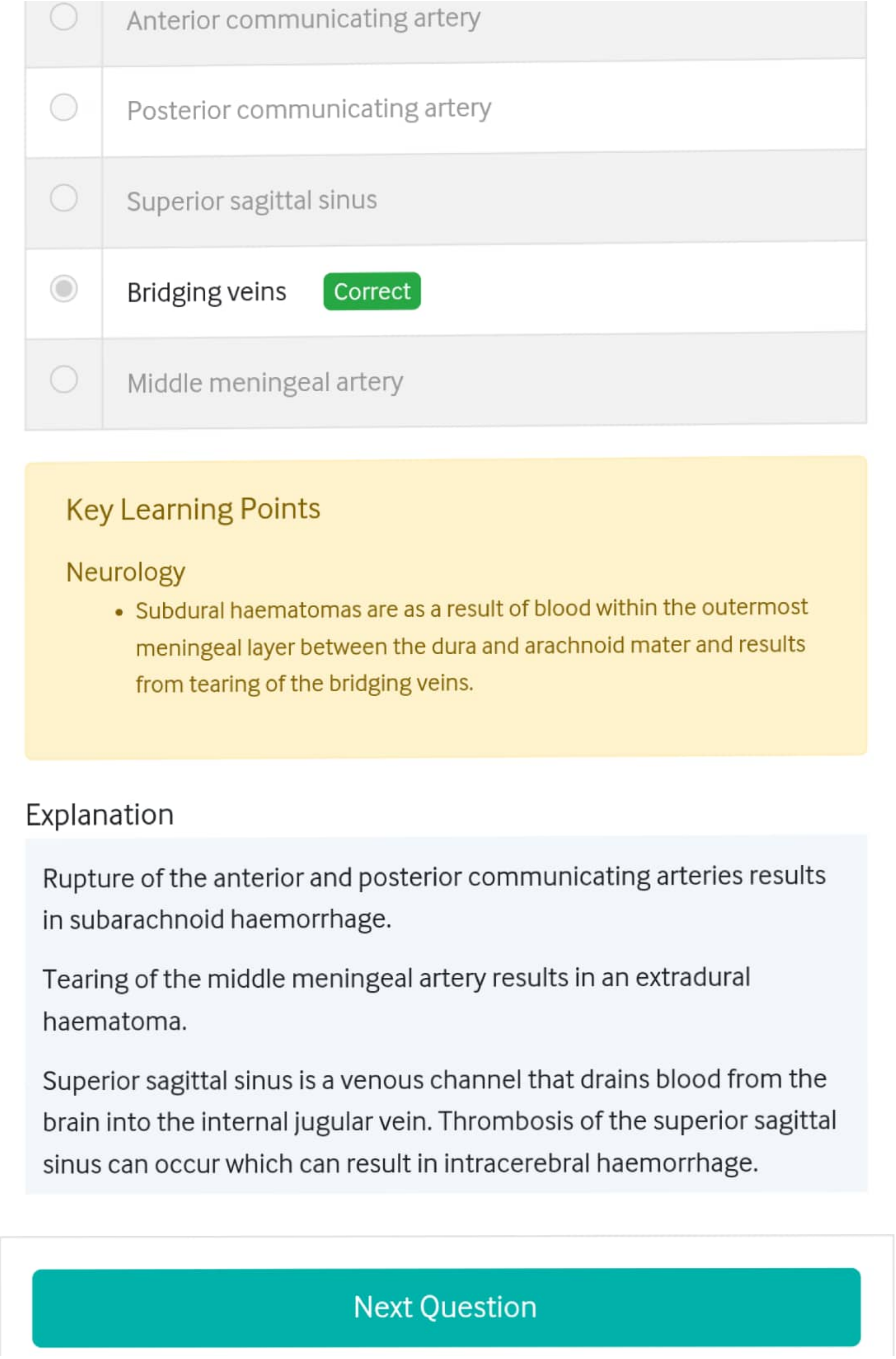
○ Posterior communicating artery

○ Superior sagittal sinus

○ Bridging veins

○ Middle meningeal artery

Submit Answer



○ Anterior communicating artery

○ Posterior communicating artery

○ Superior sagittal sinus

● Bridging veins **Correct**

○ Middle meningeal artery

Key Learning Points

Neurology

- Subdural haematomas are as a result of blood within the outermost meningeal layer between the dura and arachnoid mater and results from tearing of the bridging veins.

Explanation

Rupture of the anterior and posterior communicating arteries results in subarachnoid haemorrhage.

Tearing of the middle meningeal artery results in an extradural haematoma.

Superior sagittal sinus is a venous channel that drains blood from the brain into the internal jugular vein. Thrombosis of the superior sagittal sinus can occur which can result in intracerebral haemorrhage.

A 60-year-old man diagnosed with Alzheimer's disease was reviewed in memory clinic.

In the past three months his cognitive function had declined. His wife, who had accompanied him, expressed that he was having difficulties carrying out simple tasks such as dressing and eating. She had also noticed a paucity of speech, but felt that his general personality remained intact. He had also suffered bouts of nausea and diarrhoea.

He had been commenced on donepezil 10mg/day six months ago and for the first three months had responded to treatment. He had no other past medical history and took no other regular medication. His wife was his primary carer.

On examination he had normal demeanour and interacted well throughout the examination. He scored 11/30 on mini-mental state examination with deficiencies in recall, visuospatial tasks, language and executive function. He exhibited marked dyspraxia on testing alternating hand movements. Examination of the cranial nerves appeared normal. Examination of the peripheral nervous system revealed brisk lower limb reflexes with extensor plantar responses.

Given the above account, how would you manage this patient?

(Please select 1 option)

○ Switch to rivastigmine

○ Withdraw donepezil and consider memantine

○ Switch to galantamine

○ Remain on the same dose of donepezil and add in an anti-emetic

○ Increase the dose of donepezil

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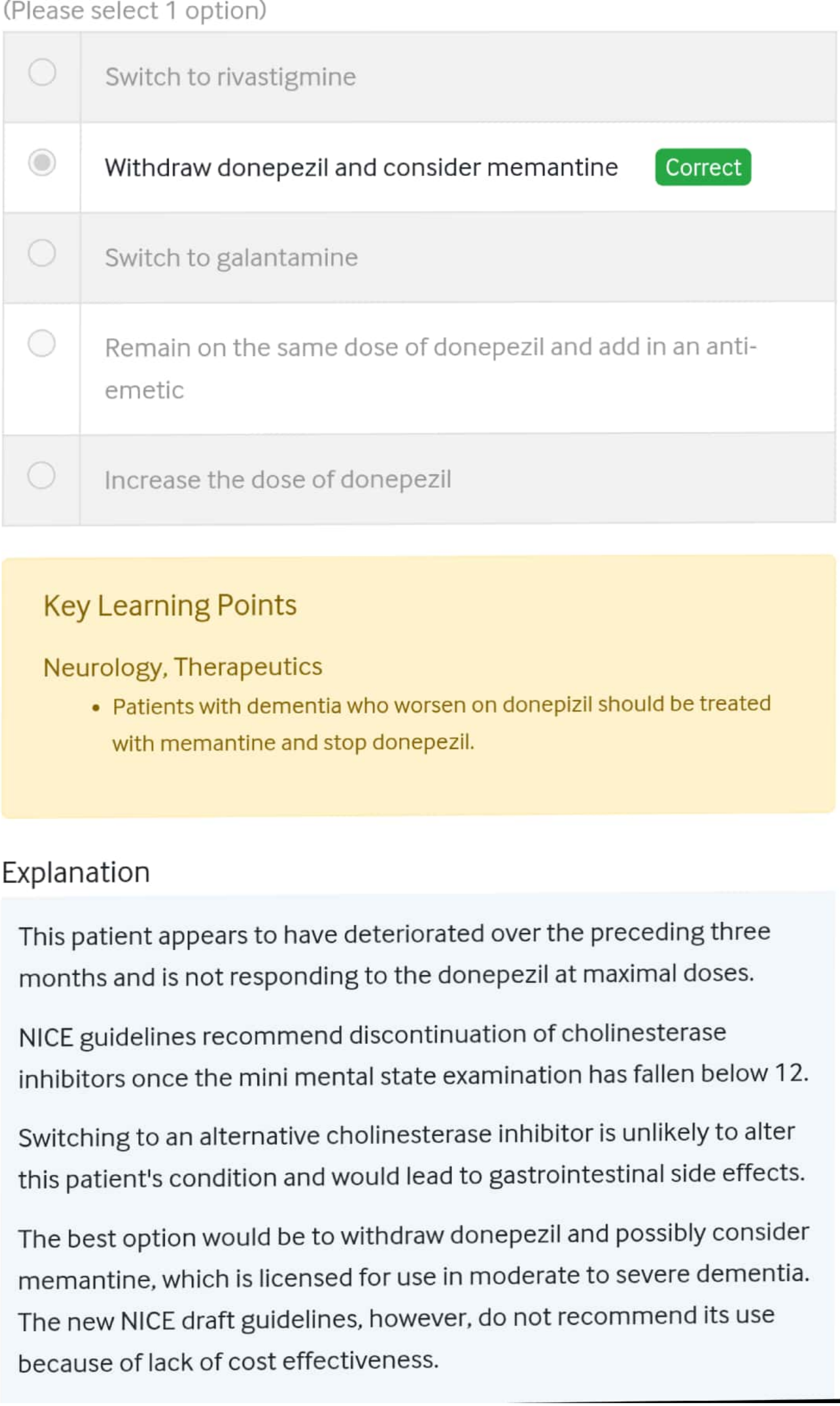


(Please select 1 option)

○ Switch to rivastigmine

● Withdraw donepezil and consider memantine Correct

○ Switch to galantamine

○ Remain on the same dose of donepezil and add in an anti-emetic

○ Increase the dose of donepezil

Key Learning Points

Neurology, Therapeutics

- Patients with dementia who worsen on donepizil should be treated with memantine and stop donepezil.

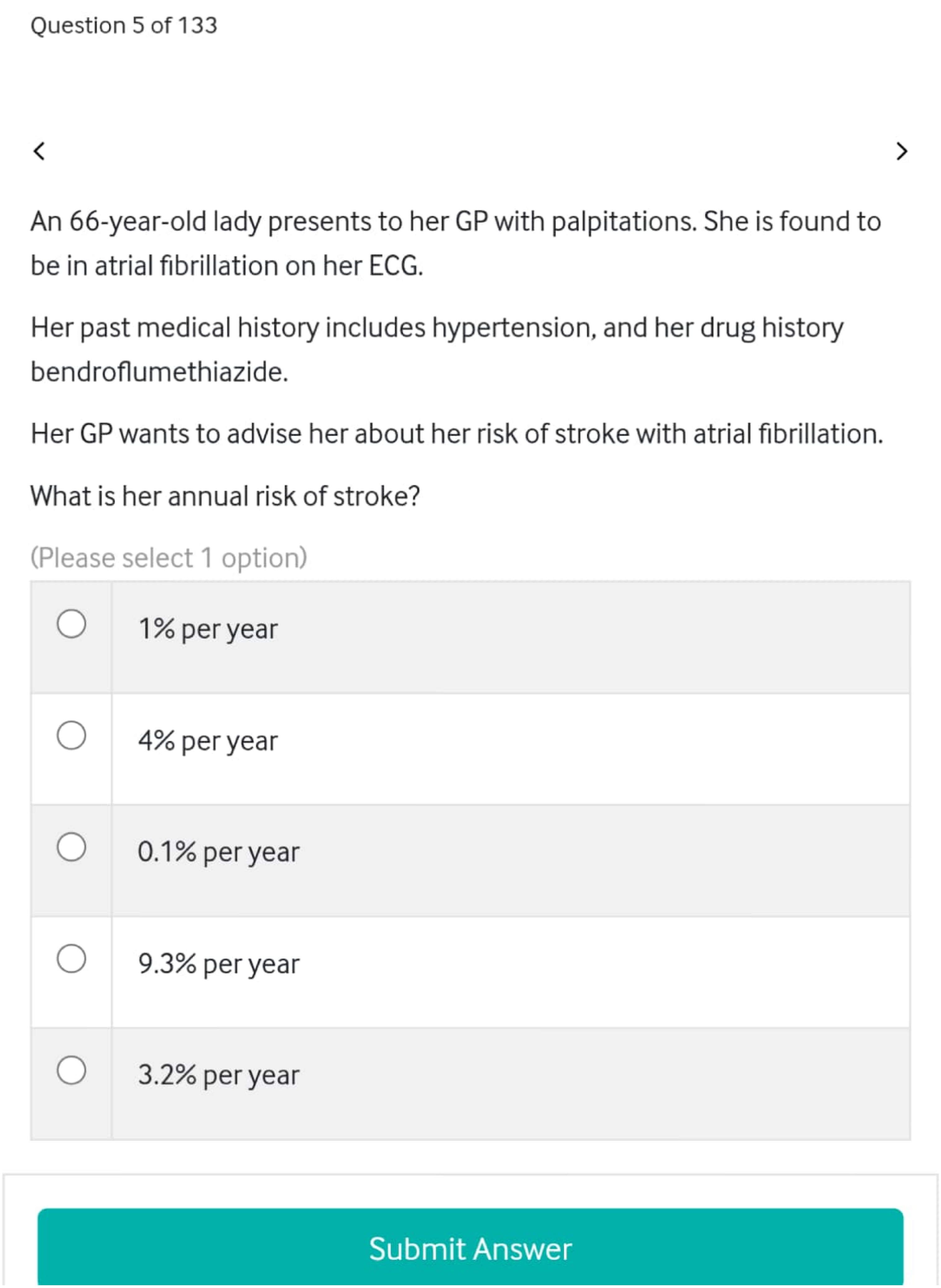
Explanation

This patient appears to have deteriorated over the preceding three months and is not responding to the donepezil at maximal doses.

NICE guidelines recommend discontinuation of cholinesterase inhibitors once the mini mental state examination has fallen below 12.

Switching to an alternative cholinesterase inhibitor is unlikely to alter this patient's condition and would lead to gastrointestinal side effects.

The best option would be to withdraw donepezil and possibly consider memantine, which is licensed for use in moderate to severe dementia. The new NICE draft guidelines, however, do not recommend its use because of lack of cost effectiveness.



Question 5 of 133

An 66-year-old lady presents to her GP with palpitations. She is found to be in atrial fibrillation on her ECG.

Her past medical history includes hypertension, and her drug history bendroflumethiazide.

Her GP wants to advise her about her risk of stroke with atrial fibrillation.

What is her annual risk of stroke?

(Please select 1 option)

- ○ 1% per year
- ○ 4% per year
- ○ 0.1% per year
- ○ 9.3% per year
- ○ 3.2% per year

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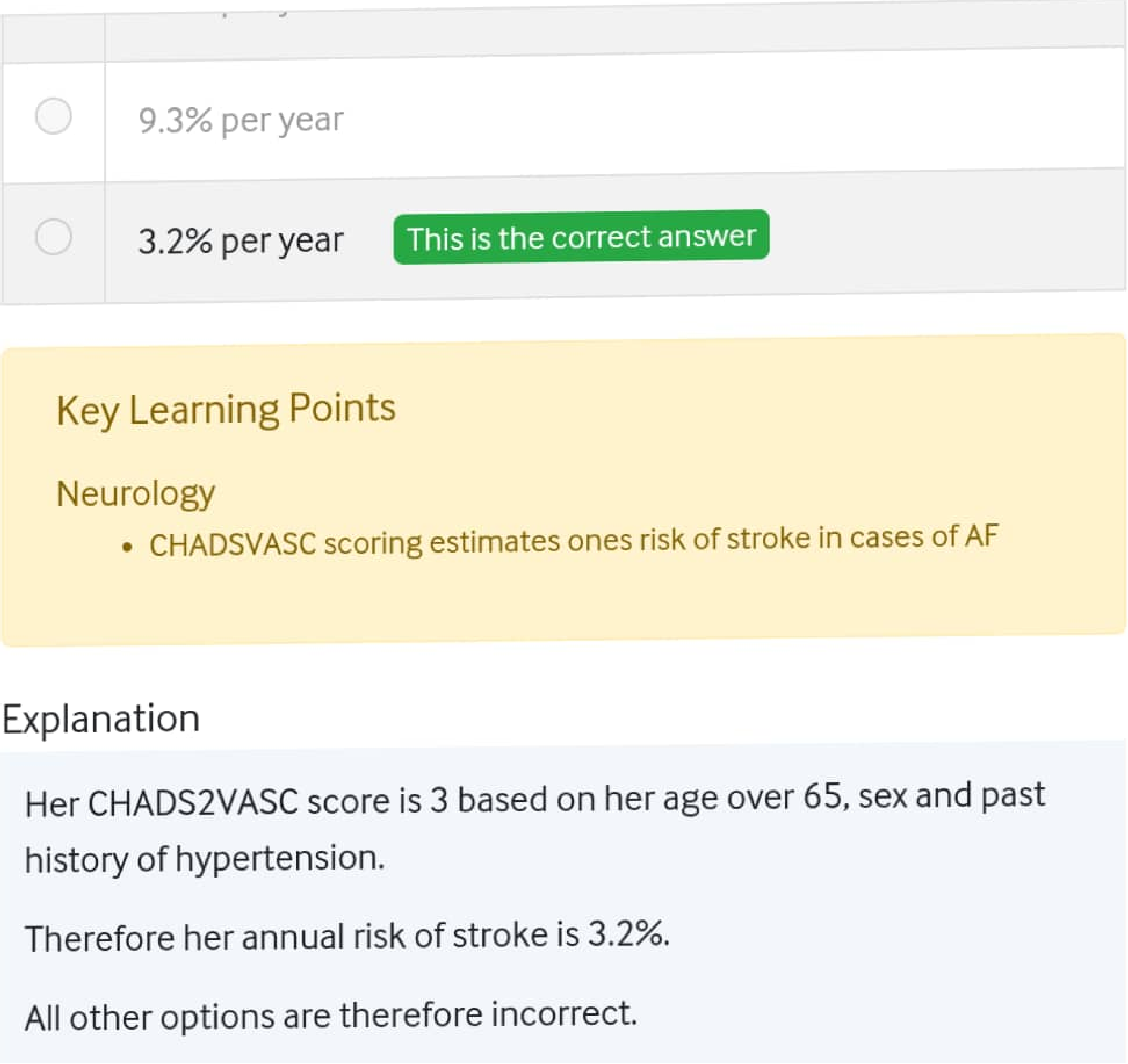

○ 9.3% per year

○ 3.2% per year

Key Learning Points

Neurology

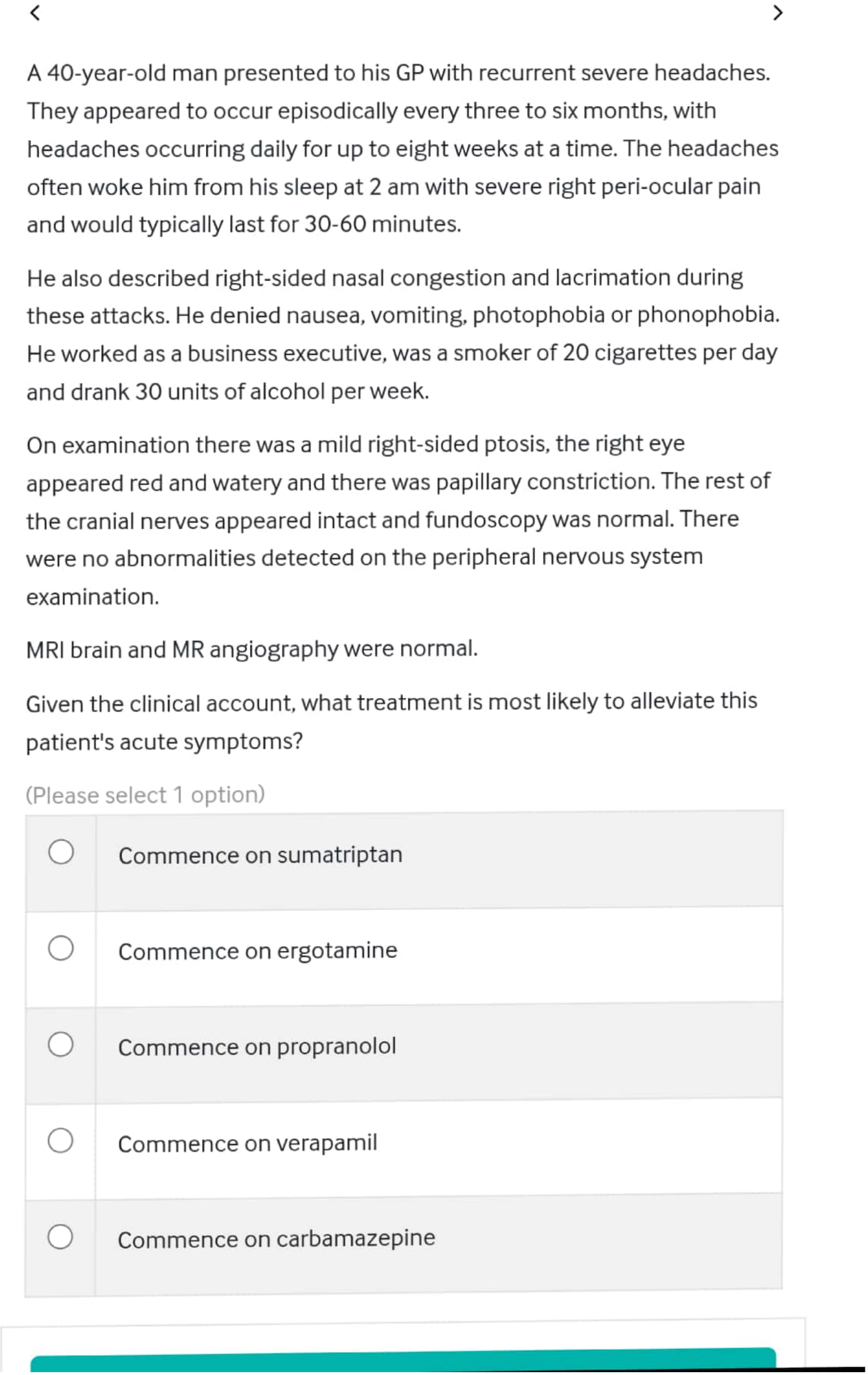
- CHADSVASC scoring estimates ones risk of stroke in cases of AF

Explanation

Her CHADS2VASC score is 3 based on her age over 65, sex and past history of hypertension.

Therefore her annual risk of stroke is 3.2%.

All other options are therefore incorrect.



A 40-year-old man presented to his GP with recurrent severe headaches. They appeared to occur episodically every three to six months, with headaches occurring daily for up to eight weeks at a time. The headaches often woke him from his sleep at 2 am with severe right peri-ocular pain and would typically last for 30-60 minutes.

He also described right-sided nasal congestion and lacrimation during these attacks. He denied nausea, vomiting, photophobia or phonophobia. He worked as a business executive, was a smoker of 20 cigarettes per day and drank 30 units of alcohol per week.

On examination there was a mild right-sided ptosis, the right eye appeared red and watery and there was papillary constriction. The rest of the cranial nerves appeared intact and fundoscopy was normal. There were no abnormalities detected on the peripheral nervous system examination.

MRI brain and MR angiography were normal.

Given the clinical account, what treatment is most likely to alleviate this patient's acute symptoms?

(Please select 1 option)

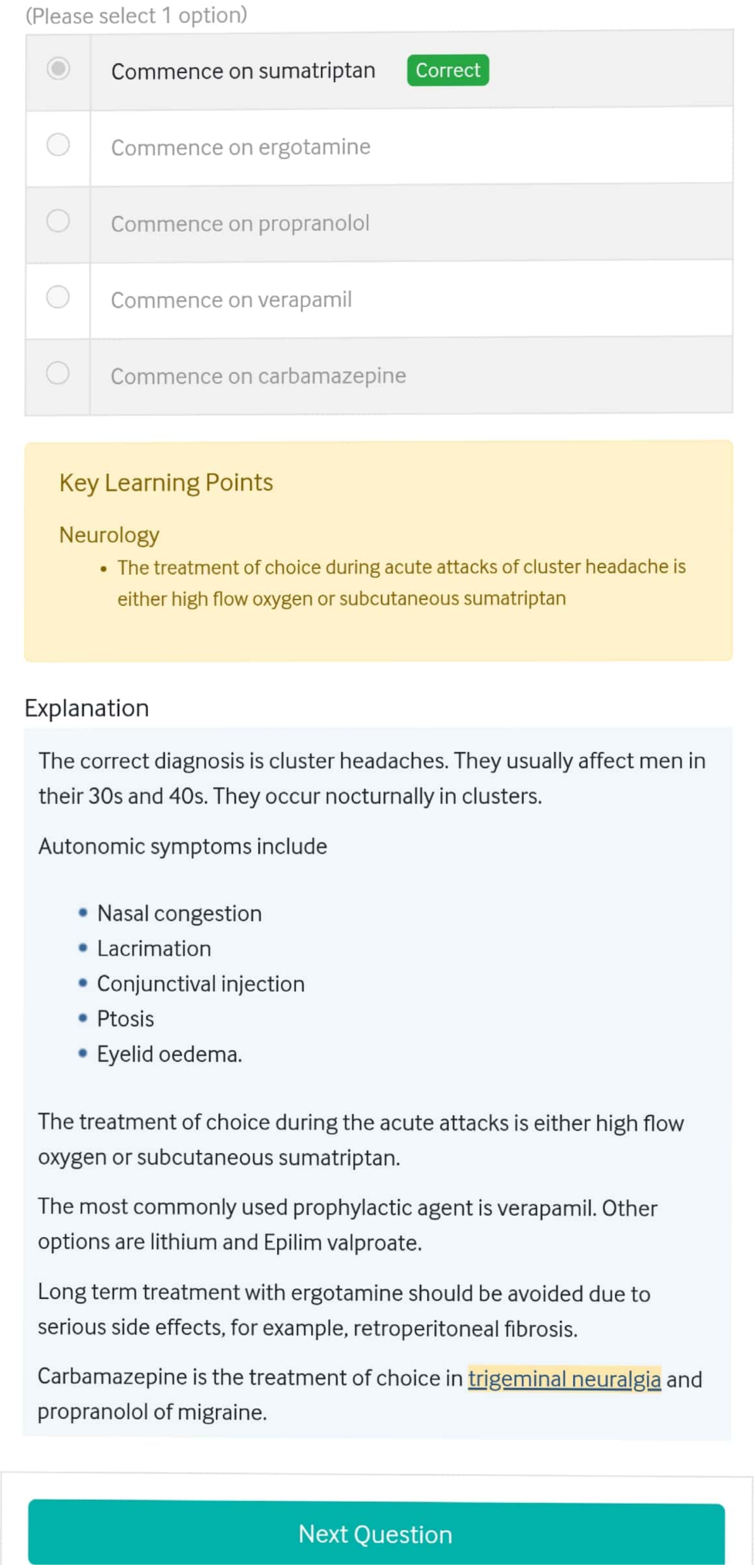
○ Commence on sumatriptan

○ Commence on ergotamine

○ Commence on propranolol

○ Commence on verapamil

○ Commence on carbamazepine



(Please select 1 option)

● Commence on sumatriptan **Correct**

○ Commence on ergotamine

○ Commence on propranolol

○ Commence on verapamil

○ Commence on carbamazepine

Key Learning Points

Neurology

- The treatment of choice during acute attacks of cluster headache is either high flow oxygen or subcutaneous sumatriptan

Explanation

The correct diagnosis is cluster headaches. They usually affect men in their 30s and 40s. They occur nocturnally in clusters.

Autonomic symptoms include

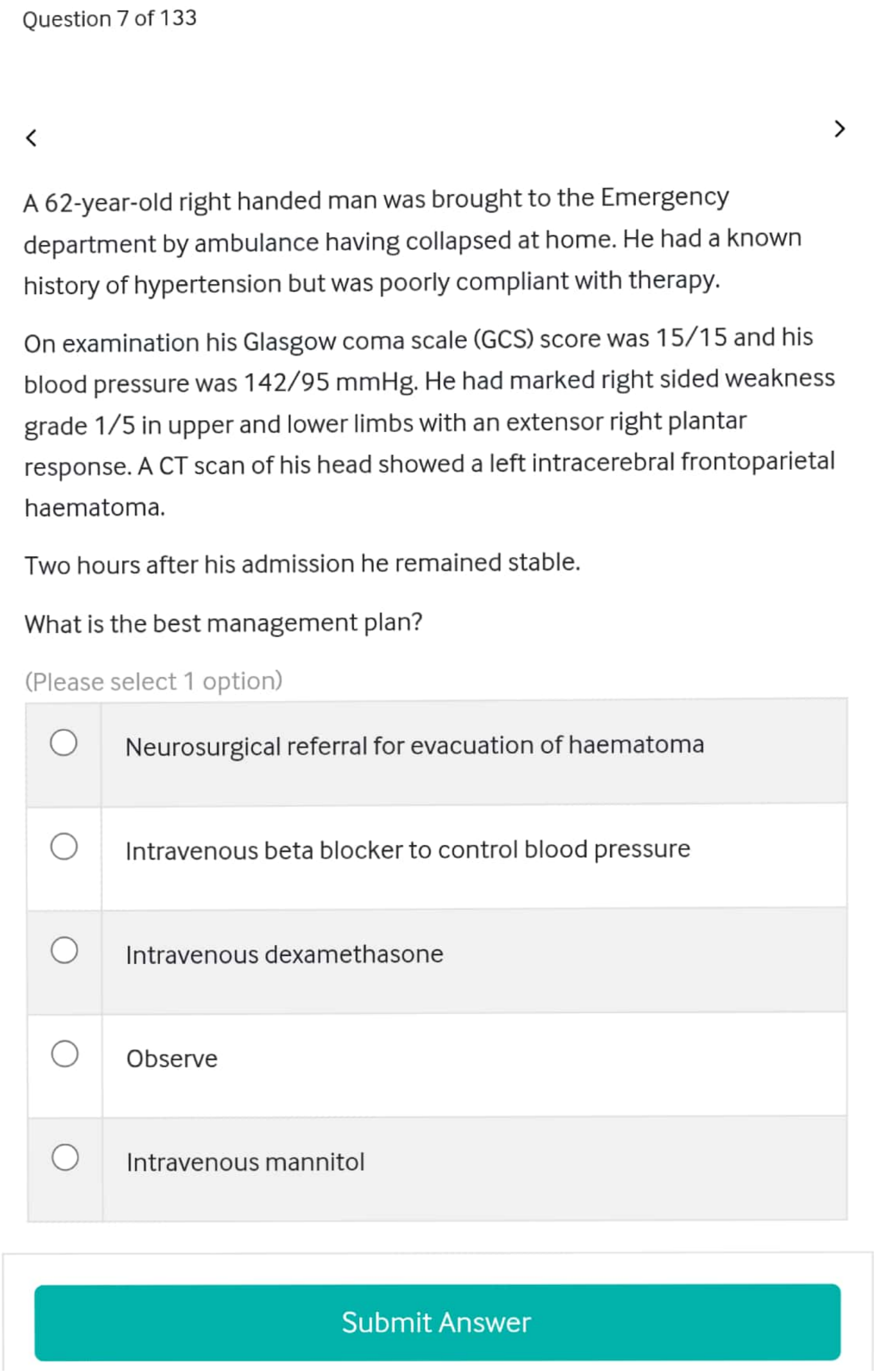
- Nasal congestion
- Lacrimation
- Conjunctival injection
- Ptosis
- Eyelid oedema.

The treatment of choice during the acute attacks is either high flow oxygen or subcutaneous sumatriptan.

The most commonly used prophylactic agent is verapamil. Other options are lithium and Epilim valproate.

Long term treatment with ergotamine should be avoided due to serious side effects, for example, retroperitoneal fibrosis.

Carbamazepine is the treatment of choice in trigeminal neuralgia and propranolol of migraine.



Question 7 of 133

A 62-year-old right handed man was brought to the Emergency department by ambulance having collapsed at home. He had a known history of hypertension but was poorly compliant with therapy.

On examination his Glasgow coma scale (GCS) score was 15/15 and his blood pressure was 142/95 mmHg. He had marked right sided weakness grade 1/5 in upper and lower limbs with an extensor right plantar response. A CT scan of his head showed a left intracerebral frontoparietal haematoma.

Two hours after his admission he remained stable.

What is the best management plan?

(Please select 1 option)

○ Neurosurgical referral for evacuation of haematoma

○ Intravenous beta blocker to control blood pressure

○ Intravenous dexamethasone

○ Observe

○ Intravenous mannitol

○ Observe **This is the correct answer**

○ Intravenous mannitol

Key Learning Points

Neurology

- Guidelines on criteria for CT head post injury should be used and followed with all patients who have had head trauma.

Explanation

There is no evidence of raised intracranial pressure (ICP) and therefore measures to reduce ICP (mannitol, sedation, assisted hyperventilation) are not appropriate currently.

In patients with an intracerebral haemorrhage, intervention to control the blood pressure is required only when the systolic BP exceeds 170 mmHg. However, the INTERACT2 trial involving 2839 patients, looked at intensive reduction of blood pressure in these patients to less than 140 mmHg. This did not significantly affect rates of death or major disability, but did significantly improve overal health-related quality of life.

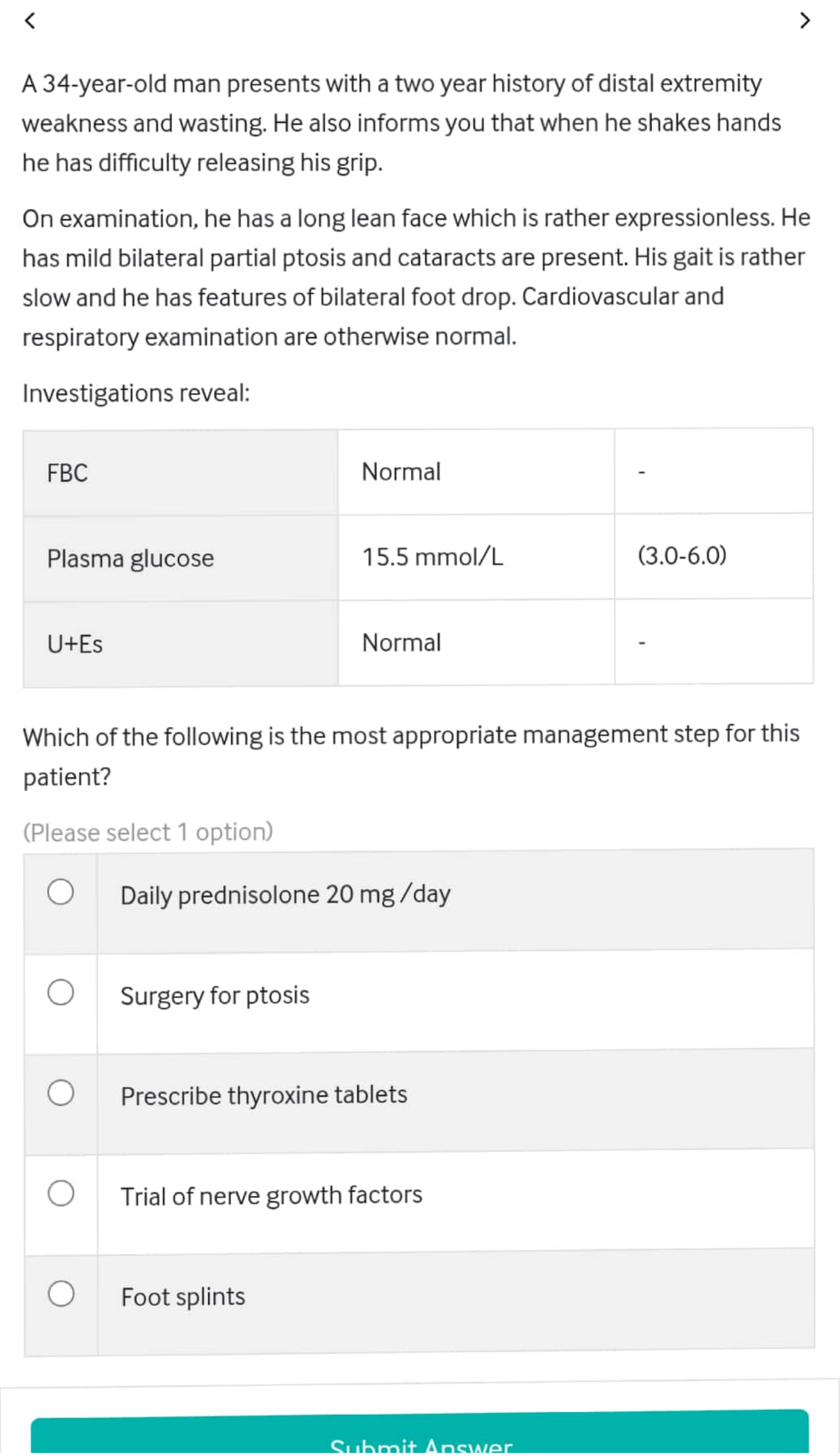
Though it may be pertinent to discuss the case with the Neurosurgical team, there is no need for their intervention at this stage. Intervention is required only when the patient has an intermediate/reducing GCS or when there is a cerebellar haemorrhage.

The patient is stable with a normal GCS.

Observation is all that is required currently.

Next Question

Go to Summary



A 34-year-old man presents with a two year history of distal extremity weakness and wasting. He also informs you that when he shakes hands he has difficulty releasing his grip.

On examination, he has a long lean face which is rather expressionless. He has mild bilateral partial ptosis and cataracts are present. His gait is rather slow and he has features of bilateral foot drop. Cardiovascular and respiratory examination are otherwise normal.

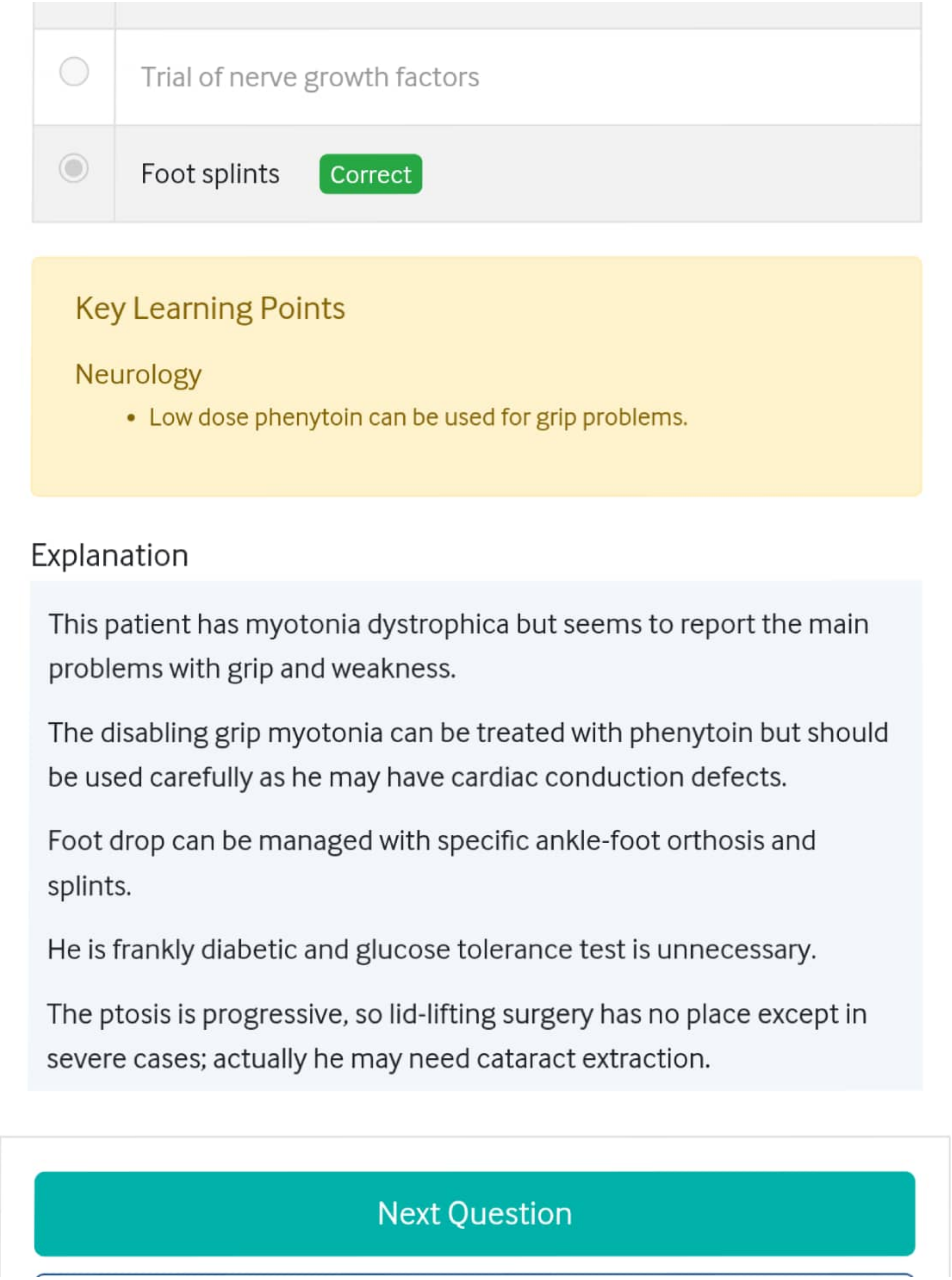
Investigations reveal:

FBC	Normal	-
Plasma glucose	15.5 mmol/L	(3.0-6.0)
U+Es	Normal	-

Which of the following is the most appropriate management step for this patient?

(Please select 1 option)

- ○ Daily prednisolone 20 mg /day
- ○ Surgery for ptosis
- ○ Prescribe thyroxine tablets
- ○ Trial of nerve growth factors
- ○ Foot splints



○	Trial of nerve growth factors
●	Foot splints **Correct**

Key Learning Points

Neurology

- Low dose phenytoin can be used for grip problems.

Explanation

This patient has myotonia dystrophica but seems to report the main problems with grip and weakness.

The disabling grip myotonia can be treated with phenytoin but should be used carefully as he may have cardiac conduction defects.

Foot drop can be managed with specific ankle-foot orthosis and splints.

He is frankly diabetic and glucose tolerance test is unnecessary.

The ptosis is progressive, so lid-lifting surgery has no place except in severe cases; actually he may need cataract extraction.

An 80-year-old man presented to his GP having developed uncontrollable flinging movements of his right arm and leg in the last few days. He had been previously well prior to the event.

The movements were irregular involving the proximal limb muscles and did not follow a particular pattern. They occurred several times a minute and had led to several falls. He had a past medical history of hypertension and ischaemic heart disease and took regular ramipril and aspirin.

He was a smoker of 10 cigarettes per day and did not drink any alcohol. There was no family history of neurological disease.

On examination he was alert and orientated, but had experienced several episodes of flinging proximal movements of his right upper and lower limb, which made examination difficult. Tone, power and reflexes all appeared normal and there were no obvious cranial nerve abnormalities. General examination revealed a blood pressure of 140/90 mmHg, pulse of 78/min and regular and heart sounds were normal.

Investigations revealed:

Haemoglobin	142 g/L	(130-180)
White cell count	6.7×10^9/L	(4-11)
Platelets	235×10^9/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	4.5 mmol/L	(3.5-4.9)
Serum urea	3.6 mmol/L	(2.5-7.5)
Serum creatinine	98 µmol/L	(60-110)
Fasting plasma glucose	8.9 mmol/L	(3-6)
Serum cholesterol	6.2 mmol/L	(< 5.2)

Given the history and above findings, where is the probable location of the lesion?

(Please select 1 option)

○ Putamen

○ Subthalamic nucleus

○ Caudate nucleus

○ Globus pallidus

○ Substantia nigra

Submit Answer

● Subthalamic nucleus **Correct**

○ Caudate nucleus

○ Globus pallidus

○ Substantia nigra

Key Learning Points

Neurology

- Knowledge of classical presentations of stroke based on an understanding of functional neuroanatomy

Explanation

The patient presents with a history in keeping with hemiballism.

The presence of severe flinging movements affecting proximal muscles and following no particular pattern is typical for hemiballism.

The site of the lesion is in the contralateral subthalamic nucleus, infarction being the commonest cause.

The patient has several arteriosclerotic risk factors including

- Hypertension
- Hyperlipidaemia
- Ischaemic heart disease and
- Diabetes.

Usually the flinging movements stop spontaneously in the next four to eight weeks and tetrabenazine is the treatment of choice.

Bilateral ballismus is rare and implicates a metabolic cause, usually non-ketotic hyperosmolar coma.

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The nurses on the neurology ward asked the senior house officer to review a patient who had developed a fever.

The patient was a 42-year-old man who had been on the ward for several months and had a long term urinary catheter in situ. The patient's neurological status was unchanged since admission, but he was not able to give any history.

On examination, the patient was lying in bed and was awake. He was febrile 37.5°C. He appeared comfortable with a respiratory rate of 12 per minute and pulse 75 beats per minute, regular. Blood pressure 120/75 mmHg.

His pupils were reactive to light, but the patient could not track a moving object - eye movements appeared to be random. He did not respond to verbal commands, though he could move all four limbs spontaneously but with no clear purpose. He did not respond to any verbal or tactile stimuli but pressure on the nail bed elicited a flexor response. Both plantar responses were extensor. The patient made moaning noises but there was no coherent speech. On occasions the patient laughed spontaneously - but not for any specific reason. The nurses reported that he had a normal sleep cycle and that he was fed by a gastrostomy feeding tube.

Urinalysis showed the presence of white cells, protein and nitrites and microscopy revealed Gram negative rods.

What is the neurological diagnosis?

(Please select 1 option)

○ Locked-in syndrome

○ Akinetic mutism

○ Coma

○ Permanent vegetative state

○ Brain death

Submit Answer

●	Permanent vegetative state **Correct**
○	Brain death

Key Learning Points

Neurology

- Patients that are inattentive and unaware of their surrounding with preserved brain stem functions is suggestive of permanent vegatative state.

Explanation

Permanent vegetative state (PVS) is defined as a state of 'wakefulness without awareness'.

The patient breathes spontaneously without mechanical support, is haemodynamically stable and has cycles of eye closure and opening that resemble a normal sleeping pattern but the patient is inattentive and unaware of his/her surroundings. Patients may have spontaneous movements (moaning, grunting, teeth grinding, roving eye movements) and may also smile, laugh and cry without any apparent reason. Although there may be eye movement, the eyes do not track a moving object. Patients may respond to painful stimuli and may have myoclonus in response to startling stimuli. Primitive reflexes may be present. Posture may become decorticate and plantar responses are commonly extensor.

The condition typically occurs when there is irreversible damage to the cerebral hemispheres but the brain stem remains intact.

Causes include

- Head injury
- Hypoxic injury (cardiac arrest, carbon monoxide poisoning)
- Stroke
- Hypoglycaemia
- Intracranial infection
- End-stage degenerative brain disease (for example, Alzheimer's).

Locked-in syndrome is a de-afferented state in which patients are aware of themselves and their environment but are unable to respond due to loss of motor and speech function.

The cause is usually either

- An upper motor neurone lesion of the descending corticospinal tracts in the brainstem (often the pons) below the level of the oculomotor nerve nuclei, (for example, infarction, haemorrhage, tumour, demyelination, head injury, central pontine myelinolysis after hyponatraemia) or
- Widespread lower motor neurone disease (for example, polyneuropathy such as Guillain-Barre syndrome).

Clinically, the patient is unable to speak or move but patients may be able to open their eyes and may blink in an effort to communicate.

Brain death involves the irreversible loss of all brainstem function. Unlike PVS, brainstem function is lost and mechanical respiratory support is required. Brain death is, by definition, irreversible and cardiac arrest usually ensues within hours or days of the onset of brain death.

The diagnosis should be made by two physicians on at least two separate occasions 12-24 hours apart.

Clinically, the patient is unconscious. Absent brainstem function is demonstrated by pupils dilated (mid- or fully) and unreactive to light. Corneal reflex is absent. 'Doll's eye' oculocephalic and caloric reflexes are absent.

Cough, suck and gag reflexes are absent. Ventilatory reflexes are absent and there is no spontaneous respiration when the patient's ventilator is switched off for sufficient period to ensure that the pCO_2 has risen above the threshold for stimulation of respiration.

Muscle tone is flaccid and there is no spontaneous movement. In order to make the diagnosis, these findings should be consistent.

Akinetic mutism is a state of profound apathy in which there is evidence of preserved awareness and visual attention and tracking. Patients frequently appear as though they are about to speak but do not ('promise of speech').

Coma. Comatose patients are unconscious and unresponsive - unaware of themselves or their environment and cannot be roused into a state of awareness or respond to their environment. Cyclical eye opening is absent and respiratory function is usually depressed.

Next Question

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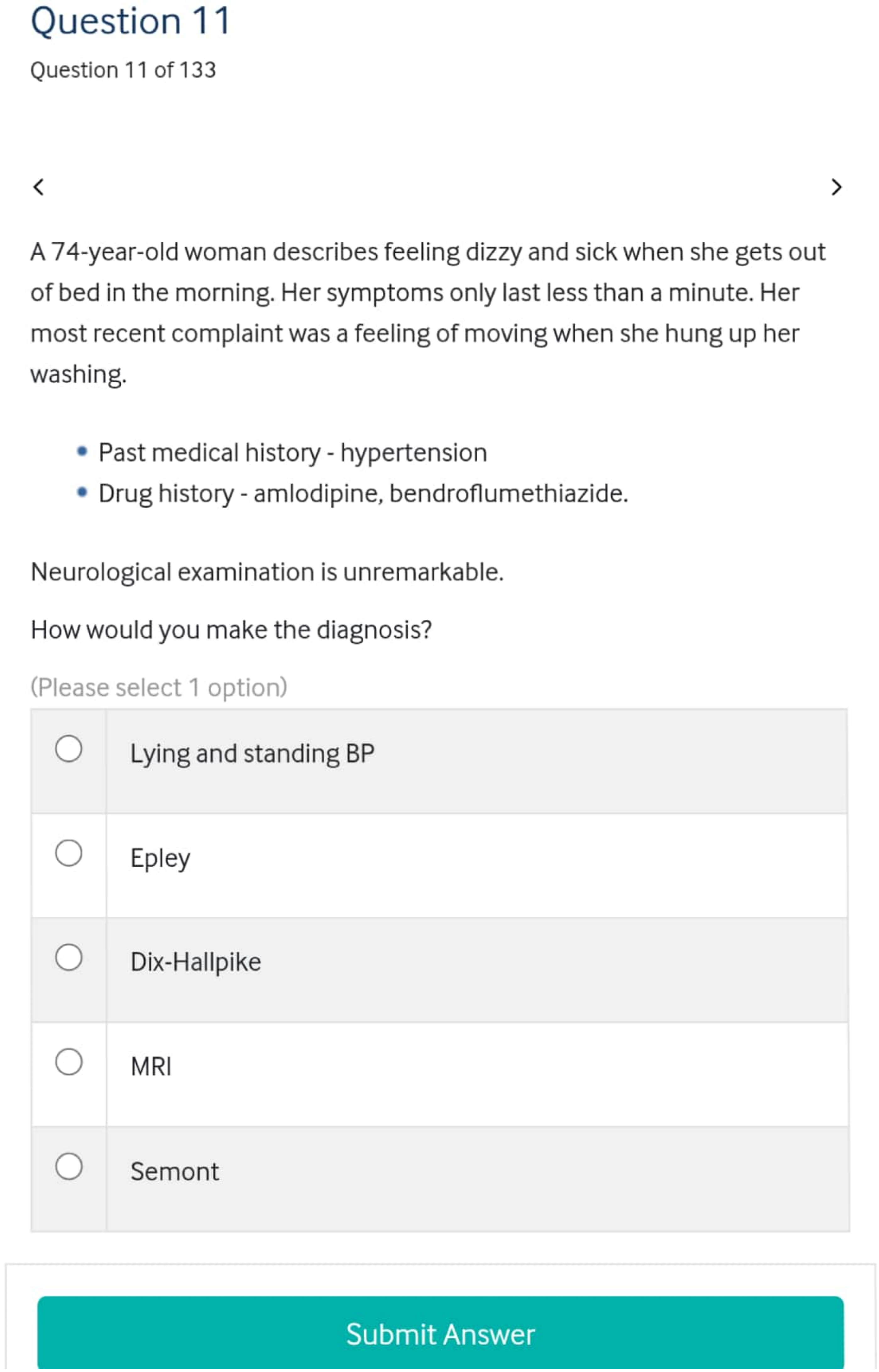

Question 11

Question 11 of 133

A 74-year-old woman describes feeling dizzy and sick when she gets out of bed in the morning. Her symptoms only last less than a minute. Her most recent complaint was a feeling of moving when she hung up her washing.

- Past medical history - hypertension
- Drug history - amlodipine, bendroflumethiazide.

Neurological examination is unremarkable.

How would you make the diagnosis?

(Please select 1 option)

○ Lying and standing BP

○ Epley

○ Dix-Hallpike

○ MRI

○ Semont

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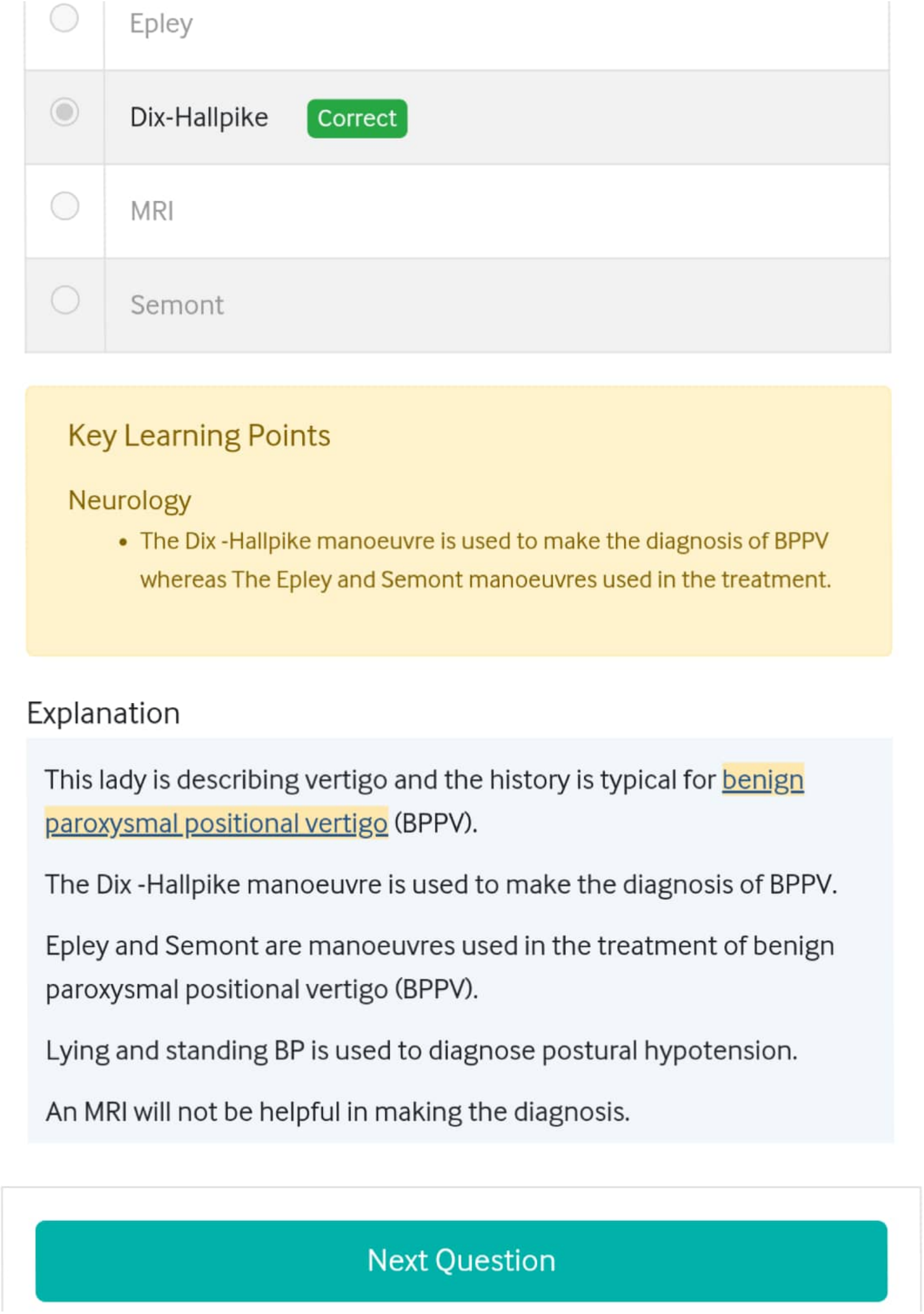


○ Epley

● Dix-Hallpike **Correct**

○ MRI

○ Semont

Key Learning Points

Neurology

- The Dix -Hallpike manoeuvre is used to make the diagnosis of BPPV whereas The Epley and Semont manoeuvres used in the treatment.

Explanation

This lady is describing vertigo and the history is typical for benign paroxysmal positional vertigo (BPPV).

The Dix -Hallpike manoeuvre is used to make the diagnosis of BPPV.

Epley and Semont are manoeuvres used in the treatment of benign paroxysmal positional vertigo (BPPV).

Lying and standing BP is used to diagnose postural hypotension.

An MRI will not be helpful in making the diagnosis.

A 60-year-old man presented to his GP with six month history of hoarse voice and choking episodes.

In the last few weeks he had also experienced pulsatile ringing in his left ear with some associated hearing loss. He denied any headache, weight loss or vomiting and had not noticed any problems with his arms or legs.

He had a past medical history of renal stones and hypertension and took allopurinol. He was a smoker of 25 cigarettes per day and did not drink alcohol.

On examination he had a husky voice with a nasal quality to his speech. There was a left Horner's syndrome, but pupils were reactive to light and ocular movements were full. Facial movements were normal and there was no obvious reduction in hearing. On examining the throat there was sluggish movement of the palate on the left and evidence of left-sided tongue wasting. There also appeared to be some difficulty in shrugging the left shoulder with weakness of chin movement to the right. The remainder of the neurological examination was normal.

Given the history and clinical findings, what is the most likely diagnosis?

(Please select 1 option)

○ Motor neuron disease

○ Hypoglossal canal syndrome

○ Nasopharyngeal carcinoma

○ Glomus jugulare tumour

○ Foramen magnum syndrome

● Glomus jugulare tumour **Correct**

○ Foramen magnum syndrome

Key Learning Points

Neurology

- Glomus jugulare tumours will cause cranial nerve palsies affecting the 9th-12th nerve.

Explanation

This patient presents with a combination of left sided 9th, 10th, 11th and 12th nerve palsies. This localises the lesion to the jugular foramen.

The patient has also developed pulsatile tinnitus and some subjective hearing loss in the left ear.

Given the symptoms and signs, the most likely cause is a glomus jugulare tumour, which is probably eroding the jugular fossa, invading the hypoglossal canal and filling the middle ear.

Foramen magnum syndrome presents with complex findings usually with a combination of lower cranial nerve dysfunction associated with long tract signs. There may be an accompanying occipital headache and neck stiffness and, on occasion, obstruction of the cerebrospinal fluid (CSF) drainage can lead to papilloedema.

Hypoglossal canal syndrome causes isolated 12th nerve palsy.

Motor neurone disease would cause a combination of upper and lower motor neurone signs with absent sensory disturbance.

With nasopharyngeal carcinoma, one would expect more localising features such as epistaxis, nasal obstruction and loss of smell. Involvement of the skull base with associated 9th, 10th, 11th and 12th nerve palsies can also occur.

A 62-year-old woman presented acutely to the Emergency department with sudden visual loss of the right eye. She had been reading at the time and suddenly noticed that she could not see the print. She denied any headache preceding the symptoms and had not noticed any weakness in her arms or legs. She had a past medical history of hypertension and took regular atenolol. She was a smoker of 20 cigarettes per day and did not drink any alcohol.

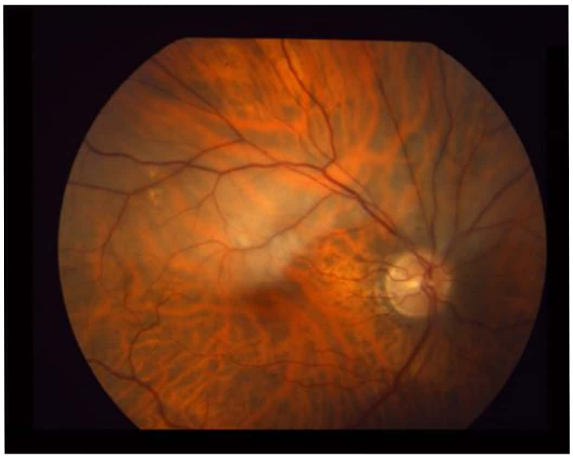
On examination she was anxious and distressed. Her blood pressure was 145/85 mmHg, pulse was 89/min and irregular and temperature was 36.7°C. On auscultation of the heart there was a mitral regurgitation murmur. On examination of the eyes the pupils were equal and reactive to light and ocular movements were full. Visual field analysis revealed a defect in the right inferior nasal field. The rest of the cranial nerves appeared normal and no abnormalities could be found in the peripheral nervous system. She had bilateral non-tender pulsatile temporal arteries.

Investigations revealed:

Haemoglobin	123 g/L	(130 - 180)
White cell count	7.8 $\times 10^9$/L	(4 - 11)
Platelets	345 $\times 10^9$/L	(150 - 400)
ESR (Westergren)	30 mm/1st hour	(0 - 30)
Serum sodium	134 mmol/L	(137 - 144)
Serum potassium	4.6 mmol/L	(3.5 - 4.9)
Serum urea	6.7 mmol/L	(2.5 - 7.5)
Serum creatinine	120 µmol/L	(60 - 110)
Serum C-reactive protein	9 IU/L	(<5)
Serum cholesterol	8.6 mmol/L	(< 5.2)

The electrocardiogram showed atrial fibrillation with a ventricular rate of 88 beats per minute.

The appearance of the retina on fundoscopy is shown below:



What is the most likely diagnosis in this patient?

(Please select 1 option)

○	Temporal arteritis
○	Cholesterol embolus of the retinal artery
○	Factitious disorder
○	Infective endocarditis
○	Parietal lobe infarct

Submit Answer

○ Temporal arteritis

● Cholesterol embolus of the retinal artery Correct

○ Factitious disorder

○ Infective endocarditis

○ Parietal lobe infarct

Key Learning Points

Neurology, Ophthalmology

- Acute monocular painless visual loss is a recognised sign of cholesterol embolism.

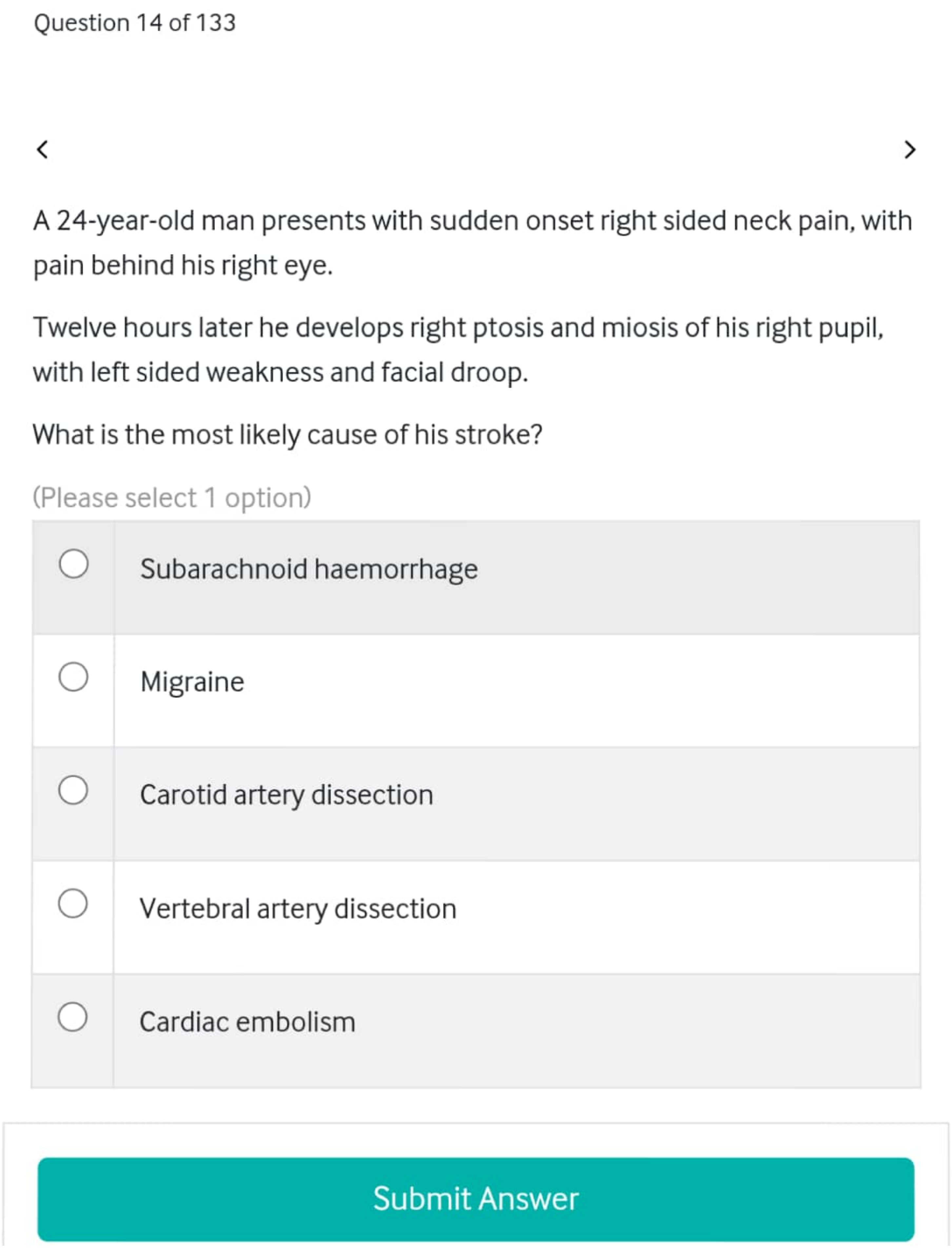
Explanation

This patient presents with a monocular visual disturbance. This places the lesion from the retina to the optic tract. She has a very specific visual disturbance affecting the inferior nasal portion of the right visual field.

Fundoscopy shows an embolus to the right superior temporal branch of the retinal artery, which would explain the clinical findings.

Temporal arteritis presents with a history of a curtain descending over the eye and, given a normal erythrocyte sedimentation rate and non-tender arteries, this diagnosis is unlikely.

Infective endocarditis is unlikely given normal inflammatory markers and haematology.



Question 14 of 133

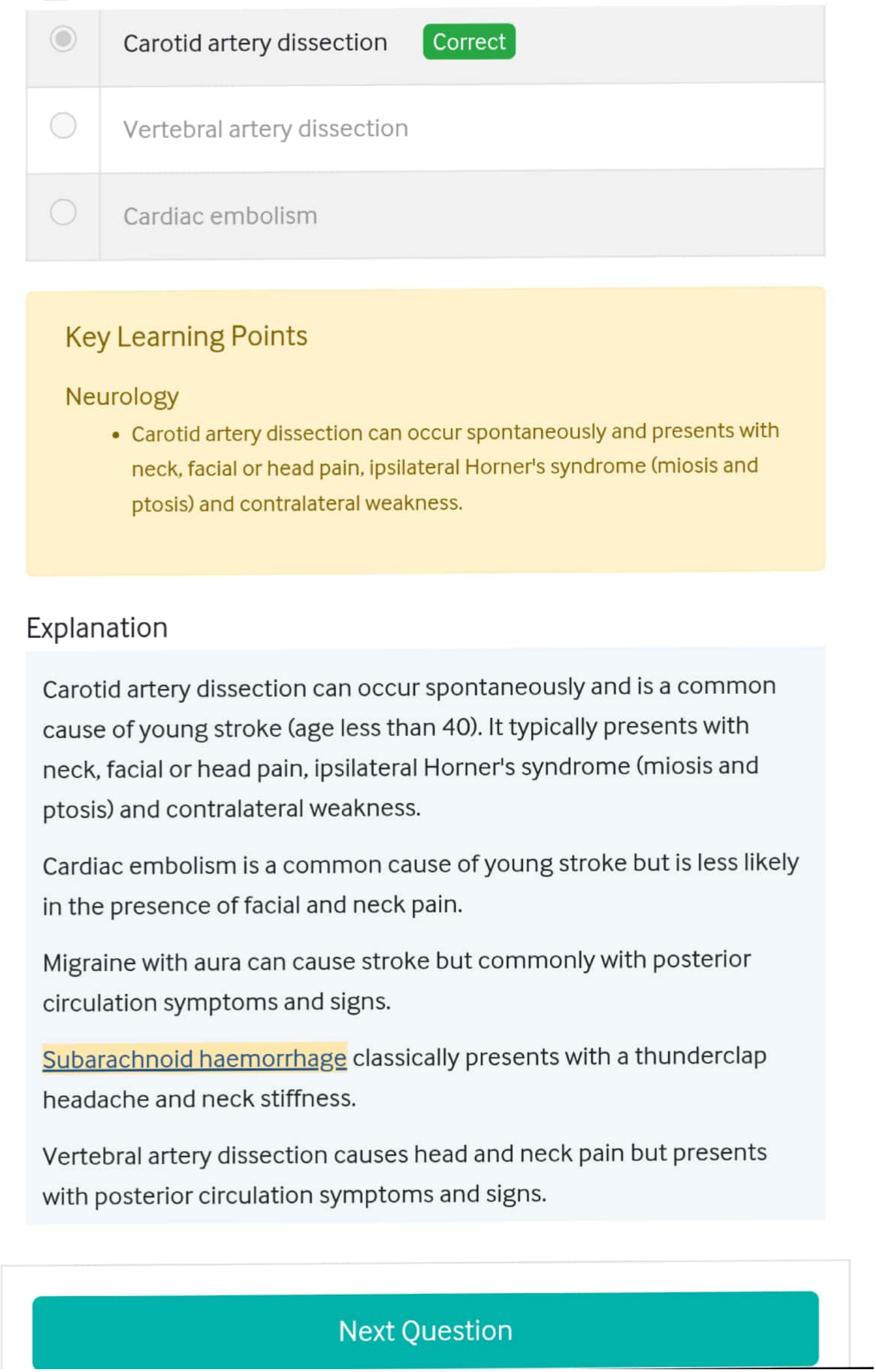
A 24-year-old man presents with sudden onset right sided neck pain, with pain behind his right eye.

Twelve hours later he develops right ptosis and miosis of his right pupil, with left sided weakness and facial droop.

What is the most likely cause of his stroke?

(Please select 1 option)

- ○ Subarachnoid haemorrhage
- ○ Migraine
- ○ Carotid artery dissection
- ○ Vertebral artery dissection
- ○ Cardiac embolism



● Carotid artery dissection **Correct**

○ Vertebral artery dissection

○ Cardiac embolism

Key Learning Points

Neurology

- Carotid artery dissection can occur spontaneously and presents with neck, facial or head pain, ipsilateral Horner's syndrome (miosis and ptosis) and contralateral weakness.

Explanation

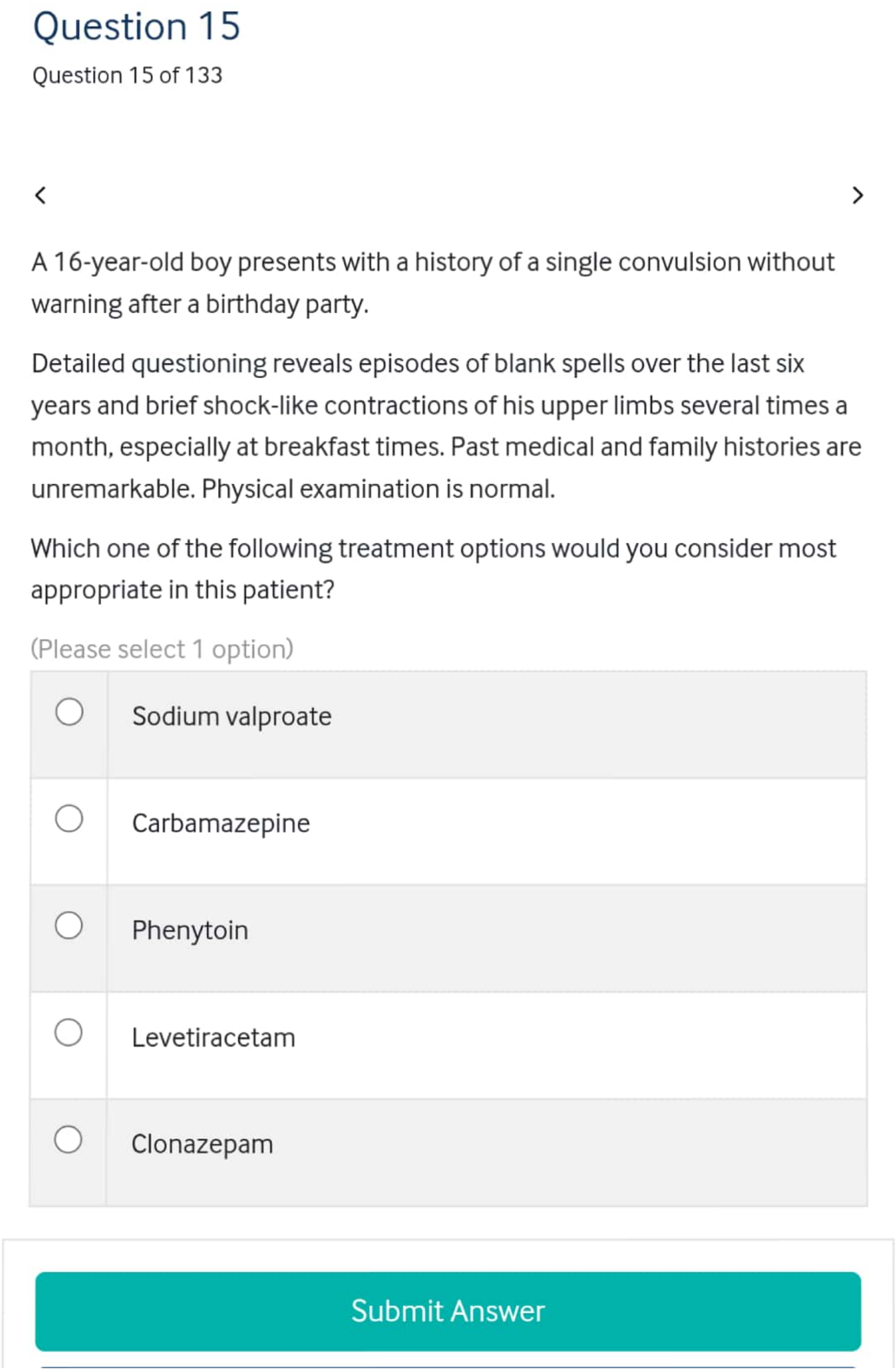
Carotid artery dissection can occur spontaneously and is a common cause of young stroke (age less than 40). It typically presents with neck, facial or head pain, ipsilateral Horner's syndrome (miosis and ptosis) and contralateral weakness.

Cardiac embolism is a common cause of young stroke but is less likely in the presence of facial and neck pain.

Migraine with aura can cause stroke but commonly with posterior circulation symptoms and signs.

Subarachnoid haemorrhage classically presents with a thunderclap headache and neck stiffness.

Vertebral artery dissection causes head and neck pain but presents with posterior circulation symptoms and signs.



Question 15

Question 15 of 133

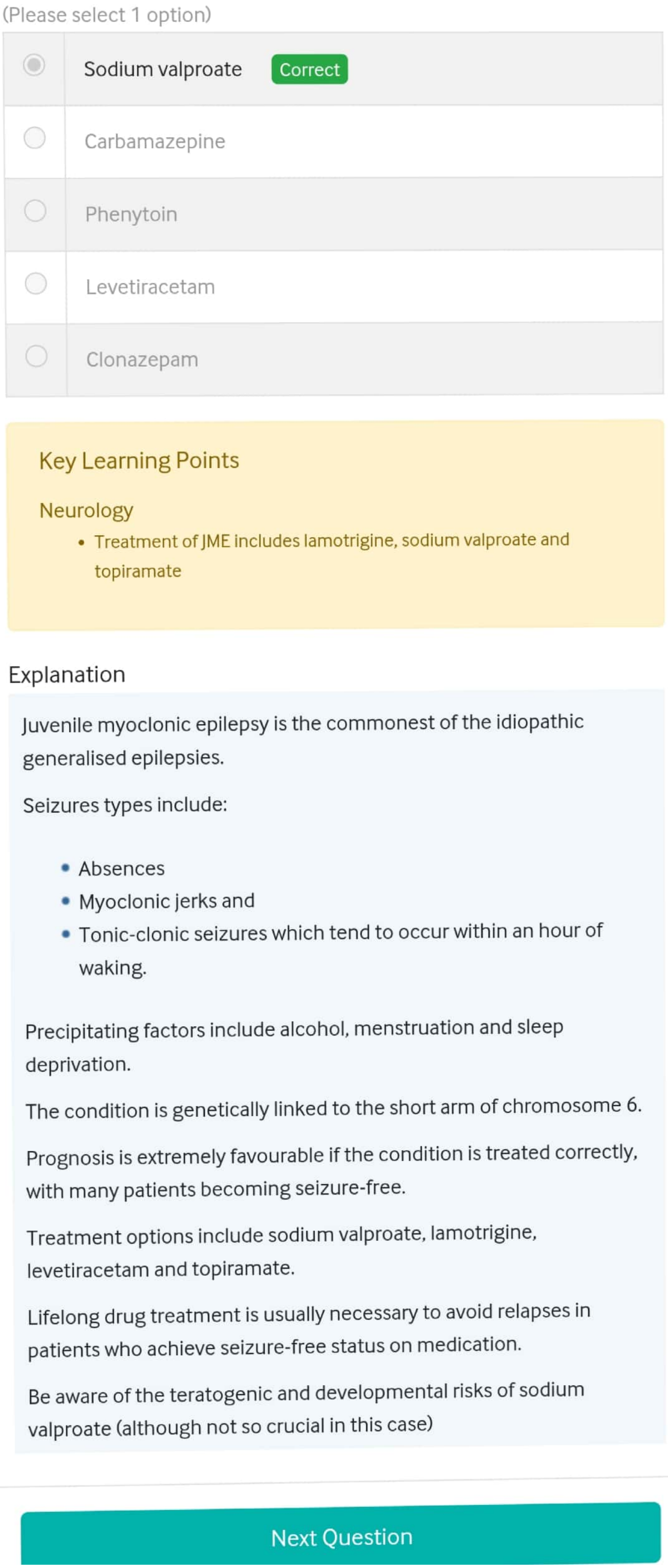
A 16-year-old boy presents with a history of a single convulsion without warning after a birthday party.

Detailed questioning reveals episodes of blank spells over the last six years and brief shock-like contractions of his upper limbs several times a month, especially at breakfast times. Past medical and family histories are unremarkable. Physical examination is normal.

Which one of the following treatment options would you consider most appropriate in this patient?

(Please select 1 option)

- ○ Sodium valproate
- ○ Carbamazepine
- ○ Phenytoin
- ○ Levetiracetam
- ○ Clonazepam



(Please select 1 option)

● Sodium valproate **Correct**

○ Carbamazepine

○ Phenytoin

○ Levetiracetam

○ Clonazepam

Key Learning Points

Neurology

- Treatment of JME includes lamotrigine, sodium valproate and topiramate

Explanation

Juvenile myoclonic epilepsy is the commonest of the idiopathic generalised epilepsies.

Seizures types include:

- Absences
- Myoclonic jerks and
- Tonic-clonic seizures which tend to occur within an hour of waking.

Precipitating factors include alcohol, menstruation and sleep deprivation.

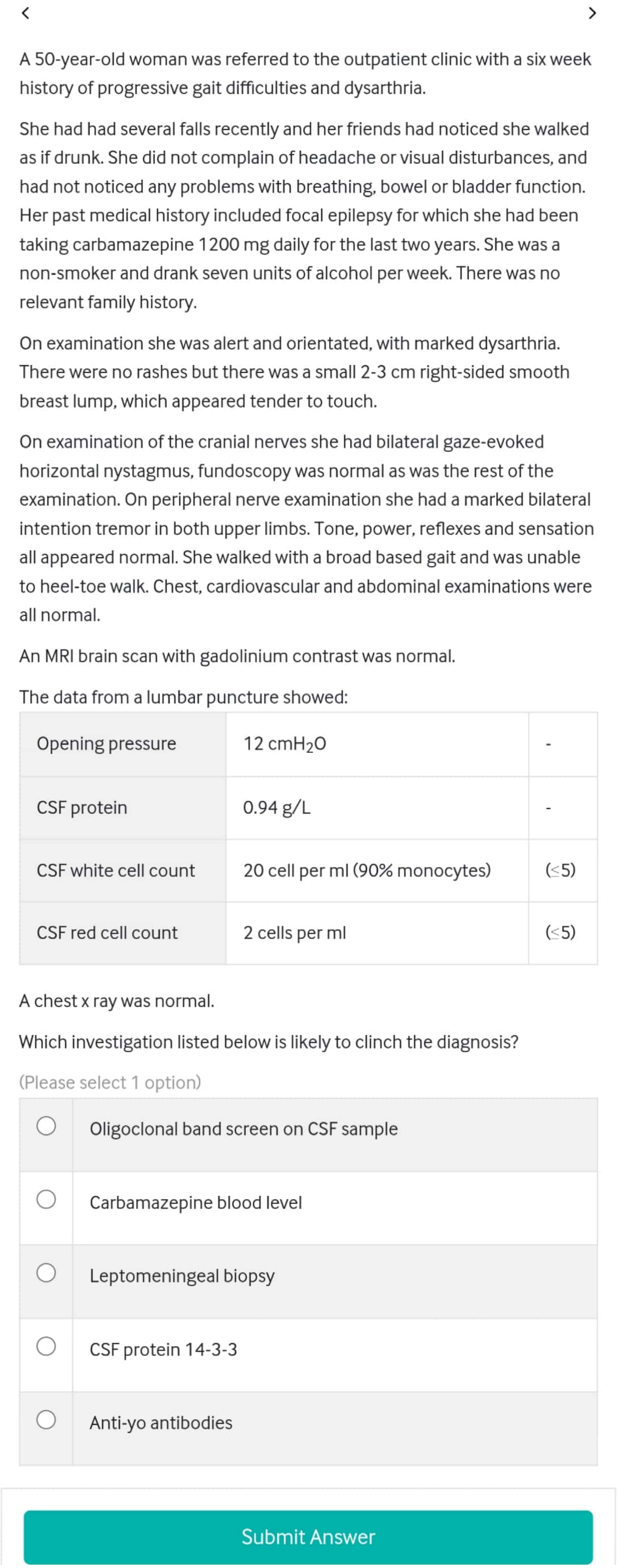
The condition is genetically linked to the short arm of chromosome 6.

Prognosis is extremely favourable if the condition is treated correctly, with many patients becoming seizure-free.

Treatment options include sodium valproate, lamotrigine, levetiracetam and topiramate.

Lifelong drug treatment is usually necessary to avoid relapses in patients who achieve seizure-free status on medication.

Be aware of the teratogenic and developmental risks of sodium valproate (although not so crucial in this case)



A 50-year-old woman was referred to the outpatient clinic with a six week history of progressive gait difficulties and dysarthria.

She had had several falls recently and her friends had noticed she walked as if drunk. She did not complain of headache or visual disturbances, and had not noticed any problems with breathing, bowel or bladder function. Her past medical history included focal epilepsy for which she had been taking carbamazepine 1200 mg daily for the last two years. She was a non-smoker and drank seven units of alcohol per week. There was no relevant family history.

On examination she was alert and orientated, with marked dysarthria. There were no rashes but there was a small 2-3 cm right-sided smooth breast lump, which appeared tender to touch.

On examination of the cranial nerves she had bilateral gaze-evoked horizontal nystagmus, fundoscopy was normal as was the rest of the examination. On peripheral nerve examination she had a marked bilateral intention tremor in both upper limbs. Tone, power, reflexes and sensation all appeared normal. She walked with a broad based gait and was unable to heel-toe walk. Chest, cardiovascular and abdominal examinations were all normal.

An MRI brain scan with gadolinium contrast was normal.

The data from a lumbar puncture showed:

Opening pressure	12 cmH_2O	-
CSF protein	0.94 g/L	-
CSF white cell count	20 cell per ml (90% monocytes)	($\leq$5)
CSF red cell count	2 cells per ml	($\leq$5)

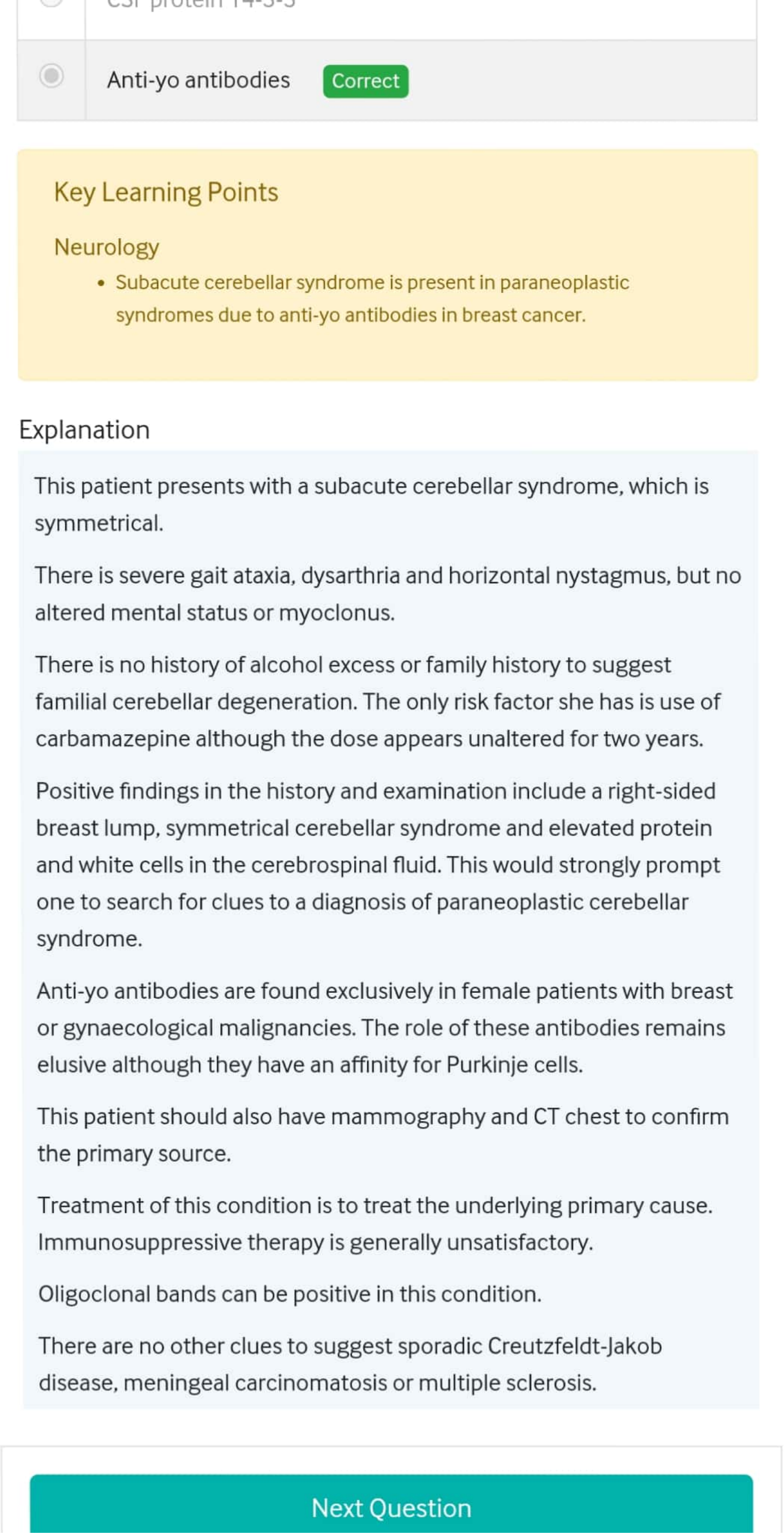
A chest x ray was normal.

Which investigation listed below is likely to clinch the diagnosis?

(Please select 1 option)

○	Oligoclonal band screen on CSF sample
○	Carbamazepine blood level
○	Leptomeningeal biopsy
○	CSF protein 14-3-3
○	Anti-yo antibodies

Submit Answer



○ CSF protein 14-3-3

● Anti-yo antibodies

Key Learning Points

Neurology

- Subacute cerebellar syndrome is present in paraneoplastic syndromes due to anti-yo antibodies in breast cancer.

Explanation

This patient presents with a subacute cerebellar syndrome, which is symmetrical.

There is severe gait ataxia, dysarthria and horizontal nystagmus, but no altered mental status or myoclonus.

There is no history of alcohol excess or family history to suggest familial cerebellar degeneration. The only risk factor she has is use of carbamazepine although the dose appears unaltered for two years.

Positive findings in the history and examination include a right-sided breast lump, symmetrical cerebellar syndrome and elevated protein and white cells in the cerebrospinal fluid. This would strongly prompt one to search for clues to a diagnosis of paraneoplastic cerebellar syndrome.

Anti-yo antibodies are found exclusively in female patients with breast or gynaecological malignancies. The role of these antibodies remains elusive although they have an affinity for Purkinje cells.

This patient should also have mammography and CT chest to confirm the primary source.

Treatment of this condition is to treat the underlying primary cause. Immunosuppressive therapy is generally unsatisfactory.

Oligoclonal bands can be positive in this condition.

There are no other clues to suggest sporadic Creutzfeldt-Jakob disease, meningeal carcinomatosis or multiple sclerosis.

A 28-year-old man was admitted to hospital with an altered conscious level and aggressive behaviour.

He had been involved in a road traffic accident three days earlier when he had been travelling at 70 mph in a residential area and had crashed into a garden wall. At the time he was taken to hospital but absconded from the Emergency department before any investigations had been conducted. He had smelt very strongly of alcohol at the time.

His family told medical staff that he took no regular medication, but did suffer from recurrent sinusitis and had taken some antibiotics recently. He was unemployed and was prone to heavy drinking. On this occasion, he was admitted with a Glasgow coma scale of 13/15 (M6, E4, V3). He appeared dishevelled, flushed and uncooperative, smelling strongly of alcohol. He had poor attention and was disorientated.

There were several lacerations to the face and a laceration over the left temporal region of the cranium. His blood pressure was 148/89 mmHg, pulse was 90/min and regular and temperature was 39°C. His heart sounds were normal and the chest was clear.

Examination of the cranial nerves revealed normal pupillary responses and extra ocular movements with no other cranial nerve abnormalities. Examination of the peripheral nervous system revealed normal tone and power, brisk reflexes which were symmetrical and flexor plantar responses. He was not cooperative for sensory examination or coordination testing.

Investigations revealed:

Haemoglobin	126 g/L	(130-180)
White cell count	18 $\times 10^9$/L	(4-11)
Platelets	456 $\times 10^9$/L	(150-400)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	3.9 mmol/L	(3.5-4.9)
Serum urea	10 mmol/L	(2.5-7.5)
Serum creatinine	108 µmol/L	(60-110)
Serum C reactive protein	450 mg/L	(<10)

A CT scan with contrast revealed a high signal oval lesion within the left frontal lobe, with some local oedema but no skull fracture. There was no contraindication to lumbar puncture.

A lumbar puncture was performed and showed:

Opening pressure	18 cmH_2O	(4 - 18)
CSF white cell count	160 cells/ml	(<5)
	90% lymphocytes	(60-70)
CSF red cell count	80 cells/ml	(<5)
CSF protein	1.1 g/L	(0.15 - 0.45)

Given the above clinical account, what is the most likely diagnosis in this patient?

(Please select 1 option)

○ Viral encephalitis

○ Arteriovenous malformation and haematoma

○ Primary cerebral lymphoma

○ Subdural empyema

○ Epidural haematoma

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○ Primary cerebral lymphoma

● Subdural empyema **Correct**

○ Epidural haematoma

Key Learning Points

Neurology

- partially treated bacterial meningitis can lead to subdural empyema.

Explanation

The clinical account and investigations are most likely to fit a diagnosis of subdural empyema with a partially treated bacterial meningoencephalitis.

The most likely origin is from chronic frontal sinus disease which is the clue in the history. He has raised inflammatory markers and white cell count, and although his cerebrospinal fluid (CSF) shows a lymphocytic pleocytosis, his protein is markedly raised and reflects partially treated bacterial meningitis.

Arteriovenous malformations normally present with seizures and a background of headaches. An epidural haematoma is associated in 75% of cases with a skull fracture, with the collection limited by firm attachments of the dura to the cranial sutures.

Primary CNS lymphoma tends to occur diffusely within white matter surrounding the ventricles. The history can be short with behavioural changes and focal epilepsy, however is less likely given the CT findings and presence of elevated inflammatory markers.

Viral encephalitis is unlikely to cause focal high signal lesions, having particular predilection to the temporal lobes.

Next Question

A 29-year-old woman attended her GP practice complaining of episodes of tunnel vision.

She tended to get these episodes whilst at work, and the tunnel vision would be preceded by dizziness and fatigue. The tunnel vision would last several minutes and was sometimes associated with dark holes within her visual range. Following an attack she would feel quite anxious and develop a headache. She had had approximately six episodes in the last month.

She had a history of type 1 diabetes which was controlled on a TDS insulin regime. She had no other medical conditions. She took co-codamol for headaches. Her mother suffered from glaucoma. She was a smoker of 20 cigarettes per day and drank approximately 20 units of alcohol, per week. She worked as a legal secretary and admitted to pressures at work.

On examination her blood pressure was 139/89 mmHg and pulse was 89 beats per minute. Heart sounds were normal and chest was clear. Fundoscopy revealed some scattered cotton-wool spots. Visual fields were normal and visual acuity was 6/6 on the left and right. There were no other cranial nerve abnormalities.

Examination of the peripheral nervous system revealed normal tone, power and reflexes. There was some diminished pin prick sensation over the dorsum and soles of both feet.

Investigations revealed:

Full blood count	Normal	-
Urea and electrolytes	Normal	-
Glucose	8.9 mmol/L	(3.0-6.0)
HbA_{1c}	74 mmol/mol	(<42)
	8.9%	(<6%)

An electrocardiogram showed a heart rate of 88 beats/min in sinus rhythm.

What is the most likely cause for this patients symptoms?

(Please select 1 option)

○ Demyelination

○ Hypoglycaemic attack

○ Glaucoma

○ Panic attack

○ Migraine aura

Submit Answer

○ Migraine aura This is the correct answer

Key Learning Points

Neurology

- Auras in migraine can be negative or positive.

Explanation

This patient is describing negative auras of dark holes and tunnel vision associated with migraine. Dizziness and fatigue are quite common prior to a migraine attack and on some occasions the aura has led to a headache.

Patients may have mixed positive and negative auras. Positive auras include bright or shimmering light or shapes at the edge of their field of vision called scintillating scotoma. They can enlarge and fill the line of vision. Other positive aura experiences are zigzag lines or stars.

Tunnel vision can occur in a panic attack but absence of anxiety, palpitations or disturbance in breathing prior to the visual symptoms makes this less likely.

In a hypoglycaemic attack there would be more systemic features such as sweating, pallor, anxiety.

She is young for the development of glaucoma which typically occurs in the fourth decade.

Fleeting visual disturbances (optic neuritis) can occur in demyelination but are much more likely to affect visual acuity, enlarge the blindspot and be associated with painful eye movements.

Next Question

Question 19

Question 19 of 133

You are referred a 35-year-old teacher who complains of episodes of severe unilateral orbital and temporal headaches lasting approximately five minutes each and occurring 20-30 times a day. The headaches have been occurring daily for more than a year with no remission. The pain is described as constant and boring.

During these episodes she also experiences mild nasal congestion and increased lacrimation.

Which of the following is the treatment of choice in this patient?

(Please select 1 option)

- ○ Propranolol
- ○ Amitriptyline
- ○ Codeine
- ○ Indomethacin
- ○ Sumatriptan

Submit Answer

○ Codeine

● Indomethacin **Correct**

○ Sumatriptan

Key Learning Points

Neurology, Therapeutics

- Chronic paroxysmal hemicrania almost invariably responds to indomethacin.

Explanation

The patient is suffering from chronic paroxysmal hemicrania which has features of cluster headaches but is associated with attacks of shorter duration and increased frequency.

In this condition, each headache can last between 3-45 minutes and occur 20-40 times per day.

In contrast, in episodic cluster headache, each headache lasts 15-180 minutes, occurs one to three times daily for four to eight weeks, with each cluster occurring several times yearly.

Chronic paroxysmal hemicrania almost invariably responds to indomethacin.

Further Reading:

Medscape: Chronic Paroxysmal Hemicrania.

Next Question

A 47-year-old man presented with a five day history of bilateral facial weakness and diplopia worse on right lateral gaze.

He could not fully close his eyes, which were becoming red and dry. He denied any recent infections or headaches, and had not noticed any difficulties with breathing or any weakness or numbness of his upper or lower limbs.

He had a past medical history of left uveitis, which was diagnosed at 18 years of age. This had been treated with topical steroids. Otherwise he was on no regular medication, was a non-smoker and drank 24 units of alcohol per week.

On examination his blood pressure was 135/80 mmHg, pulse was 75/min and regular and temperature was 37.1°C. There was a circular nodular lesion over the left shin, which appeared painful. On cranial nerve examination there was marked bilateral lower motor neuron facial weakness with an additional right VI nerve palsy. There appeared to be some fullness around the left angle of the jaw. Peripheral nervous system examination did not reveal any abnormal findings.

A CT scan of the brain was normal.

A lumbar puncture was performed and the following data were obtained:

Opening pressure	16 cmH_2O	-
CSF protein	1.5 g/L	(0.15-0.45)
CSF white cell count	125 cells per ml	(≤5)
CSF white cell differential	90% lymphocytes	-
CSF red cell count	4 cells per ml	(≤5)
CSF glucose	3.5 mmol/L	(3.3-4.4)
CSF angiotensin converting enzyme	Normal	-
CSF oligoclonal bands	Positive	-
Serum oligoclonal bands	Negative	-

What is the most likely diagnosis in this patient?

(Please select 1 option)

○ Bilateral Bell's palsy

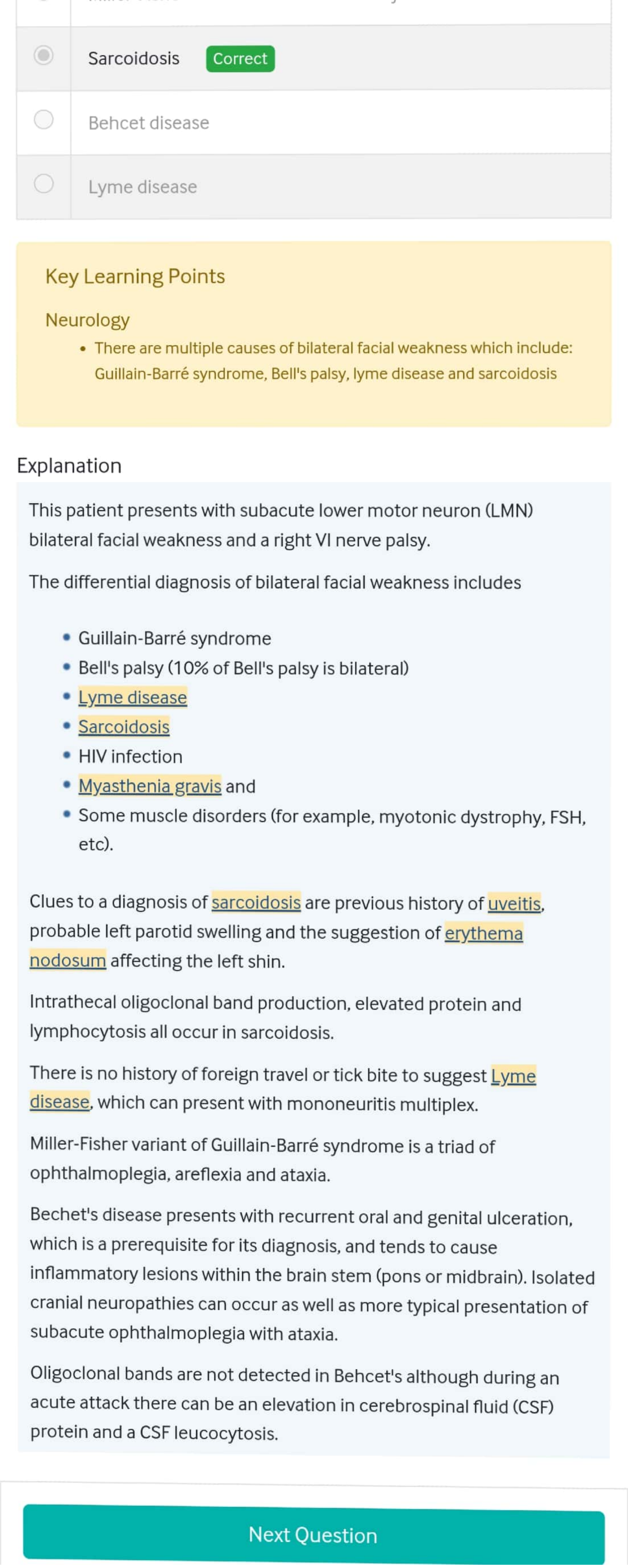
○ Miller-Fisher variant of Guillain-Barré syndrome

○ Sarcoidosis

○ Behcet disease

○ Lyme disease

Submit Answer



● Sarcoidosis **Correct**

○ Behcet disease

○ Lyme disease

Key Learning Points

Neurology

- There are multiple causes of bilateral facial weakness which include: Guillain-Barré syndrome, Bell's palsy, lyme disease and sarcoidosis

Explanation

This patient presents with subacute lower motor neuron (LMN) bilateral facial weakness and a right VI nerve palsy.

The differential diagnosis of bilateral facial weakness includes

- Guillain-Barré syndrome
- Bell's palsy (10% of Bell's palsy is bilateral)
- Lyme disease
- Sarcoidosis
- HIV infection
- Myasthenia gravis and
- Some muscle disorders (for example, myotonic dystrophy, FSH, etc).

Clues to a diagnosis of sarcoidosis are previous history of uveitis, probable left parotid swelling and the suggestion of erythema nodosum affecting the left shin.

Intrathecal oligoclonal band production, elevated protein and lymphocytosis all occur in sarcoidosis.

There is no history of foreign travel or tick bite to suggest Lyme disease, which can present with mononeuritis multiplex.

Miller-Fisher variant of Guillain-Barré syndrome is a triad of ophthalmoplegia, areflexia and ataxia.

Bechet's disease presents with recurrent oral and genital ulceration, which is a prerequisite for its diagnosis, and tends to cause inflammatory lesions within the brain stem (pons or midbrain). Isolated cranial neuropathies can occur as well as more typical presentation of subacute ophthalmoplegia with ataxia.

Oligoclonal bands are not detected in Behcet's although during an acute attack there can be an elevation in cerebrospinal fluid (CSF) protein and a CSF leucocytosis.

CamScanner المسوحة ضوئيا بـ

A 50-year-old man with learning difficulties is referred because of urinary incontinence. He lives alone. Over the last two months, he has been finding it increasingly difficult to manage simple household tasks like making meals and cleaning. He claims his "concentration is not as good as before." He had a son who died from complications of multiple sclerosis.

On examination, he is orientated in time, place and person. On memory testing, he was able to register information but was only able to recall not more than three items out of six after five minutes. Long term memory was patchy. On testing serial 3s, he reached 25 after 30 seconds. His thought content was normal, but affect was labile. He was unable to interpret the true meaning of a proverb.

Cranial nerve examination was unremarkable, except for horizontal nystagmus on lateral gaze bilaterally. Examination of the limbs revealed past-pointing in the upper limbs, difficulty with heel-to-toe walking in the lower limbs, brisk deep tendon reflexes and extensor plantar responses. Palmomental reflexes were present bilaterally.

What is the likely diagnosis?

(Please select 1 option)

○ Alzheimer's disease

○ Depression

○ Creutzfeldt-Jakob disease

○ Lewy body dementia

○ Korsakoff's amnesic syndrome

○ Depression

○ Creutzfeldt-Jakob disease

○ Lewy body dementia

○ Korsakoff's amnesic syndrome **This is the correct answer**

Key Learning Points

Neurology

- Korsakoff's syndrome is associated with heavy alcohol use and is characterised by amnesia and confabulation with lack of ability to reason.

Explanation

The recent onset of memory difficulties, mild impairment of attention and concentration and the lack of ability to reason would be more in keeping with Korsakoff's amnesic syndrome than Alzheimer's disease as the memory impairment would be more severe in the latter.

In addition, the suggestion of retained features of acute Wernicke's encephalopathy, in particular ataxia and nystagmus, add support to the diagnosis of Korsakoff's amnesic syndrome.

Thiamine is given in established cases to prevent progression of deficits.

Next Question

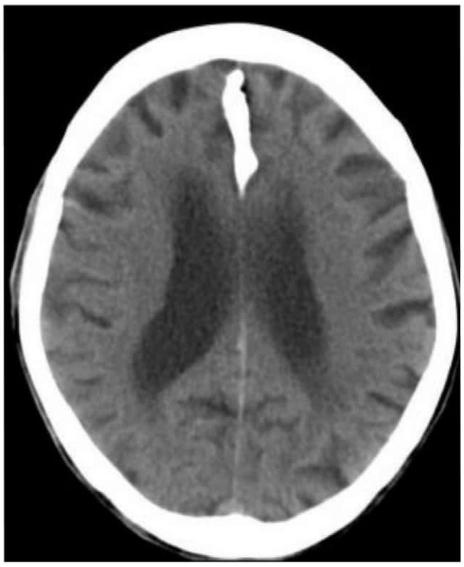
A 60-year-old man was admitted to the Emergency department with confusion and weakness of both legs.

He had developed the weakness in his legs over a period of several months and more recently had developed a generalised headache. There was no associated vomiting, visual disturbances or weakness of the upper limbs. He denied any weight loss and had not noticed any change in bowel habit or bladder function. There was no history of a recent fall or other trauma.

He had a past medical history of nasopharyngeal malignancy treated with radiotherapy two years previously. Otherwise he was well in himself and was not on any regular medication. He was an ex-smoker of 30 cigarettes per day and drank 20 units of alcohol per week.

On examination, he appeared confused with a Glasgow coma scale of 14/15 (Motor 6 eyes 4 vocal 4). His blood pressure was 140/85 mmHg, pulse 88 per minute and regular. Cranial nerve examination was normal and there were no detectable abnormalities in the upper limbs. On examination of the lower limb however, there was increased tone with bilateral pyramidal weakness and bilateral hyperreflexia and extensor plantar responses.

A CT scan of his brain was performed. An image from the non-contrasted phase of the scan is shown below:



Given the clinical history and CT scan findings what is the likely cause for this patient's symptoms?

(Please select 1 option)

○ Glioma

○ Parasaggital meningioma

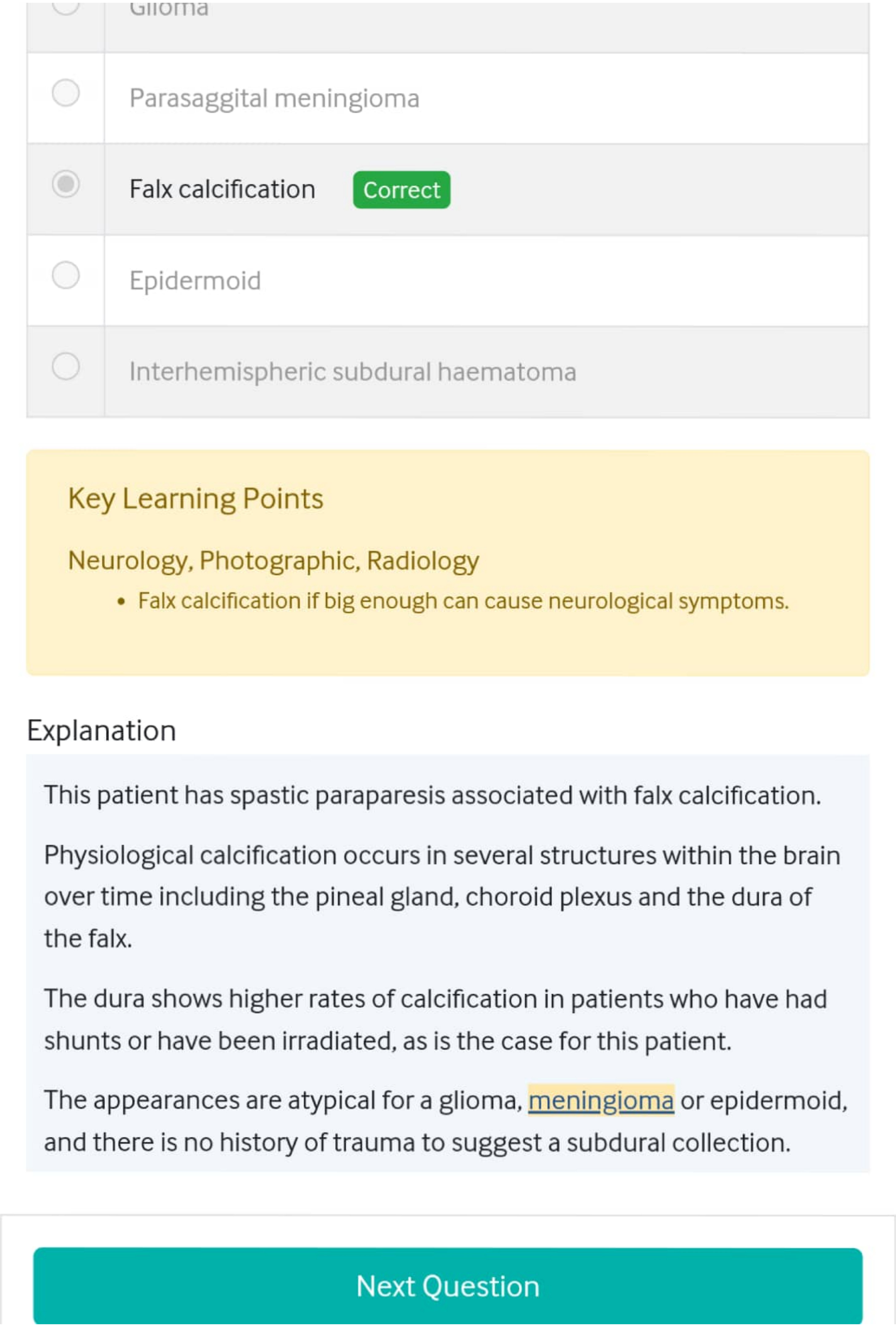
○ Falx calcification

○ Epidermoid

○ Interhemispheric subdural haematoma

Submit Answer

CamScanner المسوحة ضوئيا بـ



○ Glioma

○ Parasaggital meningioma

● Falx calcification Correct

○ Epidermoid

○ Interhemispheric subdural haematoma

Key Learning Points

Neurology, Photographic, Radiology

- Falx calcification if big enough can cause neurological symptoms.

Explanation

This patient has spastic paraparesis associated with falx calcification.

Physiological calcification occurs in several structures within the brain over time including the pineal gland, choroid plexus and the dura of the falx.

The dura shows higher rates of calcification in patients who have had shunts or have been irradiated, as is the case for this patient.

The appearances are atypical for a glioma, meningioma or epidermoid, and there is no history of trauma to suggest a subdural collection.

<   >

A 57-year-old woman presented in the Emergency department after a short episode of loss of consciousness.

Just prior to the event she had learned that her son was seriously injured in a road traffic accident.

There was no incontinence or tongue biting. The episode lasted for about a minute and she was not drowsy afterwards. She denied any headache, chest pain, breathlessness or palpitations. She has no past medical history of note and is on no medications.

On examination, pulse was 88 beats/min, BP 130/70 mmHg with no postural drop and oxygen saturation 99% on air. Cardiovascular and neurological examinations were unremarkable.

Which of the following is the most appropriate investigation to perform first?

(Please select 1 option)

- ○ ECG and cardiac monitor
- ○ Serum electrolytes and glucose
- ○ CT scan head
- ○ EEG
- ○ Ventilation-perfusion scan

Submit Answer

Which of the following is the most appropriate investigation to perform first?

(Please select 1 option)

- ○ ECG and cardiac monitor **This is the correct answer**
- ○ Serum electrolytes and glucose
- ○ CT scan head
- ● EEG **Incorrect answer selected**
- ○ Ventilation-perfusion scan

Key Learning Points

Neurology

- The ECG is a quick tool to categorise a presentation of syncope into high and low risk.

Explanation

In the acute situation the ECG is the most useful test for classifying syncopal episodes into high risk and low risk categories.

High risk cases are those of abrupt onset occurring in patients older than 55 years who have a history of heart disease or abnormal ECG.

Low risk patients are those who have no underlying diseases and a normal ECG.

Next Question

CamScanner الممسوحة ضوئيا بـ

A 30-year-old mechanic presented to the Emergency department with vertigo, dysarthria, ataxia, nausea and vomiting.

In the preceding five hours he had developed a severe occipital pain, which started whilst working under a car. He denied any hearing impairment or any visual disturbances. He had no past medical history and he did not take any prescribed medication. He was a smoker of 20 cigarettes per day and drank 20 units of alcohol per week; however he denied any illicit drug abuse. There was a family history of stroke and his mother had died of subarachnoid haemorrhage at the age of 43 years.

On examination he was pale and vomiting and complained of severe occipital head pain. Pulse was 80/min and regular, blood pressure was 120/80 mmHg and temperature was 36.5°C. Heart sounds were normal. The pupils appeared sluggishly reactive to light and fundoscopy was normal.

On cranial nerve examination, he exhibited gaze-evoked nystagmus towards the right, and had a marked dysarthria. On peripheral nervous system examination, there was an intention tremor of the right arm. Tone, power, reflexes and sensation were all normal. On lower limb examination he walked with an ataxic gait and appeared to be veering towards the right.

In view of the above findings, what is the likely diagnosis in this man?

(Please select 1 option)

○ Extra cranial vertebral artery dissection

○ Extra cranial carotid artery dissection

○ Lateral medullary syndrome

○ Subarachnoid haemorrhage

○ Basilar migraine

In view of the above findings, what is the likely diagnosis in this man?

(Please select 1 option)

● Extra cranial vertebral artery dissection **Correct**

○ Extra cranial carotid artery dissection

○ Lateral medullary syndrome

○ Subarachnoid haemorrhage

○ Basilar migraine

Key Learning Points

Neurology

- Dissection and cardioembolism are the commonest causes of stroke in young people.

Explanation

This patient presents with a classical history in keeping with extra-cranial vertebral artery dissection.

Dissection and cardioembolism are the commonest causes of stroke in young people (less than 40-years-old). The onset of severe occipital pain usually caused by trivial trauma is a typical presentation for dissection.

Symptoms in keeping with ischaemia of posterior circulation occur (cerebellar, brainstem signs).

Extra-cranial carotid artery dissection presents with haematoma in the neck and possible compression of the sympathetic chain leading to an ipsilateral Horner's syndrome and cranial neuropathies particularly cranial nerves IX, X, XI and XII.

Lateral medullary syndrome occurs as a result of posterior inferior cerebellar artery infarct. This is characterised by ipsilateral Horner's syndrome, ipsilateral reduced pain and temperature sensation of the face and other ipsilateral cranial nerve involvement and contralateral loss of pain and temperature sensation of the limbs.

Next Question

CamScanner المسوحة ضوئيا بـ

A 60-year-old man was referred to a neurologist with a three month history of progressive forgetfulness, agitation and speech problems. He had been working as a shelf stacker in a supermarket, but had recently been made redundant because he had become increasingly clumsy with items. Since then his wife had noticed him to be somewhat low in mood.

His past medical history included hypertension for which he was treated with bendroflumethiazide and hypothyroidism treated with thyroxine.

On examination his vital signs were normal. He was found to be low in mood and had a marked expressive dysphasia with a receptive component. On the mini mental state examination he scored 21/30 with deficiencies in recall, orientation and visuo-constructive tasks. There was a left palmomental reflex.

Peripheral nerve examination revealed marked generalised myoclonus and in the upper limb an intention tremor. In the lower limb, he exhibited a broad based gait but no other features. Tone, power and reflexes and sensation were all normal.

Blood tests showed:

Sodium	130 mmol/L	(137-144)
Potassium	4.0 mmol/L	(3.5-4.9)
Urea	7.2 mmol/L	(2.5-7.5)
Creatinine	154 μmol/L	(60-110)
Thyroxine	60 nmol/L	(58-178)
Thyroid stimulating hormone	8.0 mU/L	(0.4-5)
Vitamin B_{12}	250 ng/L	(160-760)
Folate	7.0 g/L	(2.0-11.0)
Haemoglobin	134 g/L	(130-180)
Mean cell volume	102 fL	(80-96)
White cell count	8.0×10^9/L	(4-11)
Platelets	250×10^9/L	(150-400)

Erythrocyte sedimentation rate: 15 mm/hr (1-20)

What is the most likely cause for this patient's symptoms?

(Please select 1 option)

○ Hypothyroidism

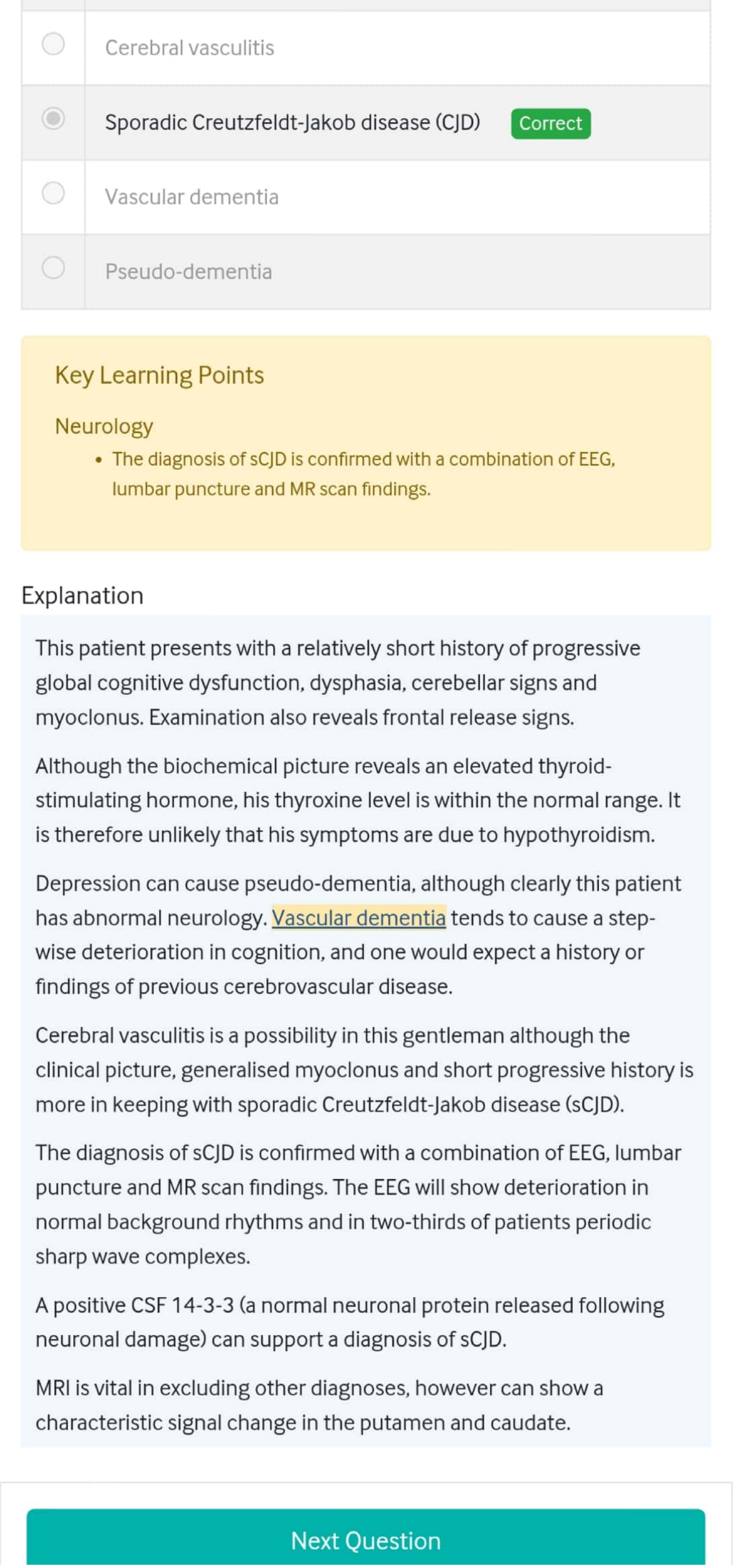
○ Cerebral vasculitis

○ Sporadic Creutzfeldt-Jakob disease (CJD)

○ Vascular dementia

○ Pseudo-dementia

Submit Answer



○ Cerebral vasculitis

● Sporadic Creutzfeldt-Jakob disease (CJD) Correct

○ Vascular dementia

○ Pseudo-dementia

Key Learning Points

Neurology

- The diagnosis of sCJD is confirmed with a combination of EEG, lumbar puncture and MR scan findings.

Explanation

This patient presents with a relatively short history of progressive global cognitive dysfunction, dysphasia, cerebellar signs and myoclonus. Examination also reveals frontal release signs.

Although the biochemical picture reveals an elevated thyroid-stimulating hormone, his thyroxine level is within the normal range. It is therefore unlikely that his symptoms are due to hypothyroidism.

Depression can cause pseudo-dementia, although clearly this patient has abnormal neurology. Vascular dementia tends to cause a step-wise deterioration in cognition, and one would expect a history or findings of previous cerebrovascular disease.

Cerebral vasculitis is a possibility in this gentleman although the clinical picture, generalised myoclonus and short progressive history is more in keeping with sporadic Creutzfeldt-Jakob disease (sCJD).

The diagnosis of sCJD is confirmed with a combination of EEG, lumbar puncture and MR scan findings. The EEG will show deterioration in normal background rhythms and in two-thirds of patients periodic sharp wave complexes.

A positive CSF 14-3-3 (a normal neuronal protein released following neuronal damage) can support a diagnosis of sCJD.

MRI is vital in excluding other diagnoses, however can show a characteristic signal change in the putamen and caudate.

A 22-year-old female was referred to a neurologist having developed involuntary movements affecting her arms and legs. She described the movements as a 'shock wave'. They were not painful and lasted seconds but appeared to occur many times throughout the day. In addition she had had notable cognitive decline and complained of poor memory.

She had a history of tonic-clonic seizures and had recently been diagnosed by her GP with impaired glucose tolerance. She was taking carbamazepine for her epilepsy. There was a family history of early onset dementia on her mother's side.

On examination she was five foot in height and appeared to have slow mentation. She scored 27/30 on a mini mental state examination, and had impaired recall.

Fundoscopy examination revealed bilateral optic atrophy. Cranial nerve examination was normal other than some left lateralisation on Weber's testing and normal bone: air conduction on the left and right.

On examination of the upper limb she had multiple lipomata affecting both arms and exhibited some brief involuntary movements. Tone and reflexes were normal, however there was some slight weakness of shoulder abduction. On examination of the lower limb, tone, power and reflexes appeared normal however she walked with a mildly ataxic gait.

Investigations revealed:

Serum sodium	145 mmol/L	(137 - 144)
Serum potassium	3.7 mmol/L	(3.5 - 4.9)
Serum urea	6.7 mmol/L	(2.5 - 7.5)
Serum creatinine	99 µmol/L	(24 - 170)
Serum glucose	6.7 mmol/L	(4-6)
Serum carbamazepine	45 µmmol/L	(Therapeutic range 34-51)

A lumbar puncture was performed. Analysis of cerebrospinal fluid showed:

CSF opening pressure	140 mmH$_2$O	(50-180)
CSF protein	0.4 g/L	(0.15-0.45)
CSF white cell count	4 per ml	($\leq$5)
CSF red cell count	2 per ml	(5)
CSF lactate	3.2 mmol/L	(1-2)
CSF oligoclonal bands	Negative	-

EEG shows generalised slow waves. MRI brain scan is normal.

Given the above findings so far, what is the likely diagnosis in this lady?

(Please select 1 option)

○ Neuronal ceroid-lipofuscinosis

○ Huntington's disease

○ New variant Creutzfeld-Jakob disease (NvCJD)

○ MERRF syndrome (myoclonic epilepsy and red ragged fibres)

○ Juvenile myoclonic epilepsy

Submit Answer

○	New variant Creutzfeld-Jakob disease (NvCJD)
○	MERRF syndrome (myoclonic epilepsy and red ragged fibres) This is the correct answer
○	Juvenile myoclonic epilepsy

Key Learning Points

Neurology

- 60% of patients with MERRF syndrome present with generalised myoclonus.

Explanation

This patient presents with a full house of symptoms and signs consistent with MERRF (myoclonic epilepsy and red ragged fibres). She has generalised myoclonus (60%), ataxia, cognitive decline and encephalopathy (EEG findings of generalised slow waves) and epilepsy. In addition she has short stature (10%), optic atrophy (20%), right sensorineural hearing loss and impaired glucose tolerance.

Huntington's disease presents in middle age with dementia and choreiform movement disorder. This patient clearly has a lot more going on than juvenile myoclonic epilepsy.

Neuronal ceroid-lipofuscinosis is a lysosomal storage disorder of lipids and typically presents in infancy and childhood. It can present with epilepsy and ataxia.

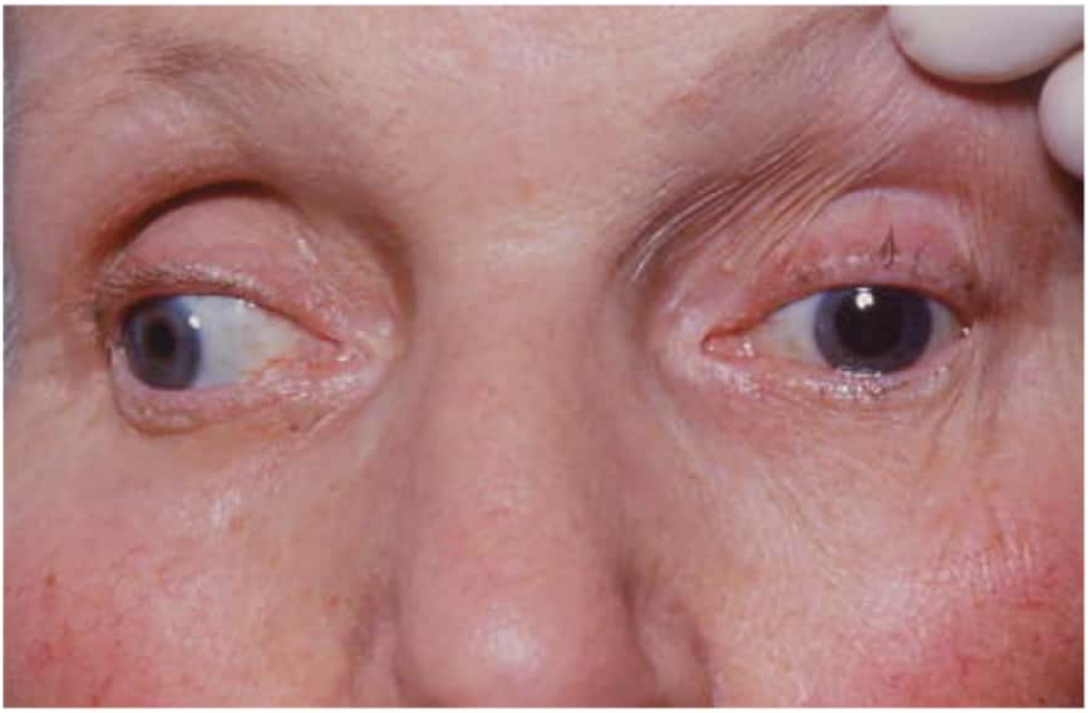
In addition these patients have lipomata, and some develop retinal degeneration.

Genetic testing is available for this condition.

NvCJD typically presents with psychiatric and sensory disturbances.

Sporadic CJD presents later in life (60s) with dementia, myoclonus and cerebellar dysfunction.

This 53-year-old man with diabetes who presents with headache and double vision was asked to look to his right.



What is the diagnosis?

(Please select 1 option)

○ Left third and seventh nerve palsies

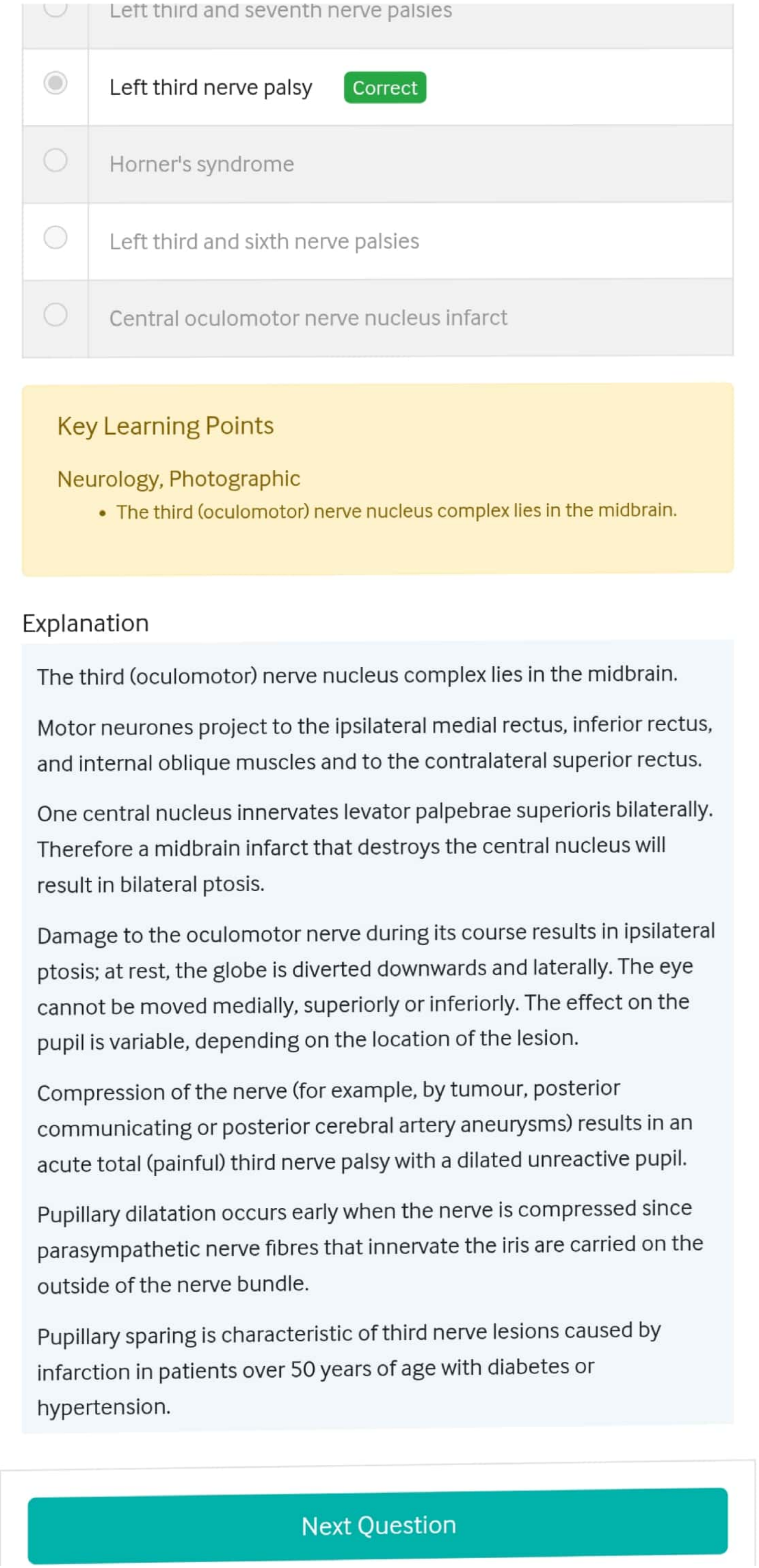
○ Left third nerve palsy

○ Horner's syndrome

○ Left third and sixth nerve palsies

○ Central oculomotor nerve nucleus infarct

Submit Answer



○ Left third and seventh nerve palsies

● Left third nerve palsy **Correct**

○ Horner's syndrome

○ Left third and sixth nerve palsies

○ Central oculomotor nerve nucleus infarct

Key Learning Points

Neurology, Photographic

- The third (oculomotor) nerve nucleus complex lies in the midbrain.

Explanation

The third (oculomotor) nerve nucleus complex lies in the midbrain.

Motor neurones project to the ipsilateral medial rectus, inferior rectus, and internal oblique muscles and to the contralateral superior rectus.

One central nucleus innervates levator palpebrae superioris bilaterally. Therefore a midbrain infarct that destroys the central nucleus will result in bilateral ptosis.

Damage to the oculomotor nerve during its course results in ipsilateral ptosis; at rest, the globe is diverted downwards and laterally. The eye cannot be moved medially, superiorly or inferiorly. The effect on the pupil is variable, depending on the location of the lesion.

Compression of the nerve (for example, by tumour, posterior communicating or posterior cerebral artery aneurysms) results in an acute total (painful) third nerve palsy with a dilated unreactive pupil.

Pupillary dilatation occurs early when the nerve is compressed since parasympathetic nerve fibres that innervate the iris are carried on the outside of the nerve bundle.

Pupillary sparing is characteristic of third nerve lesions caused by infarction in patients over 50 years of age with diabetes or hypertension.

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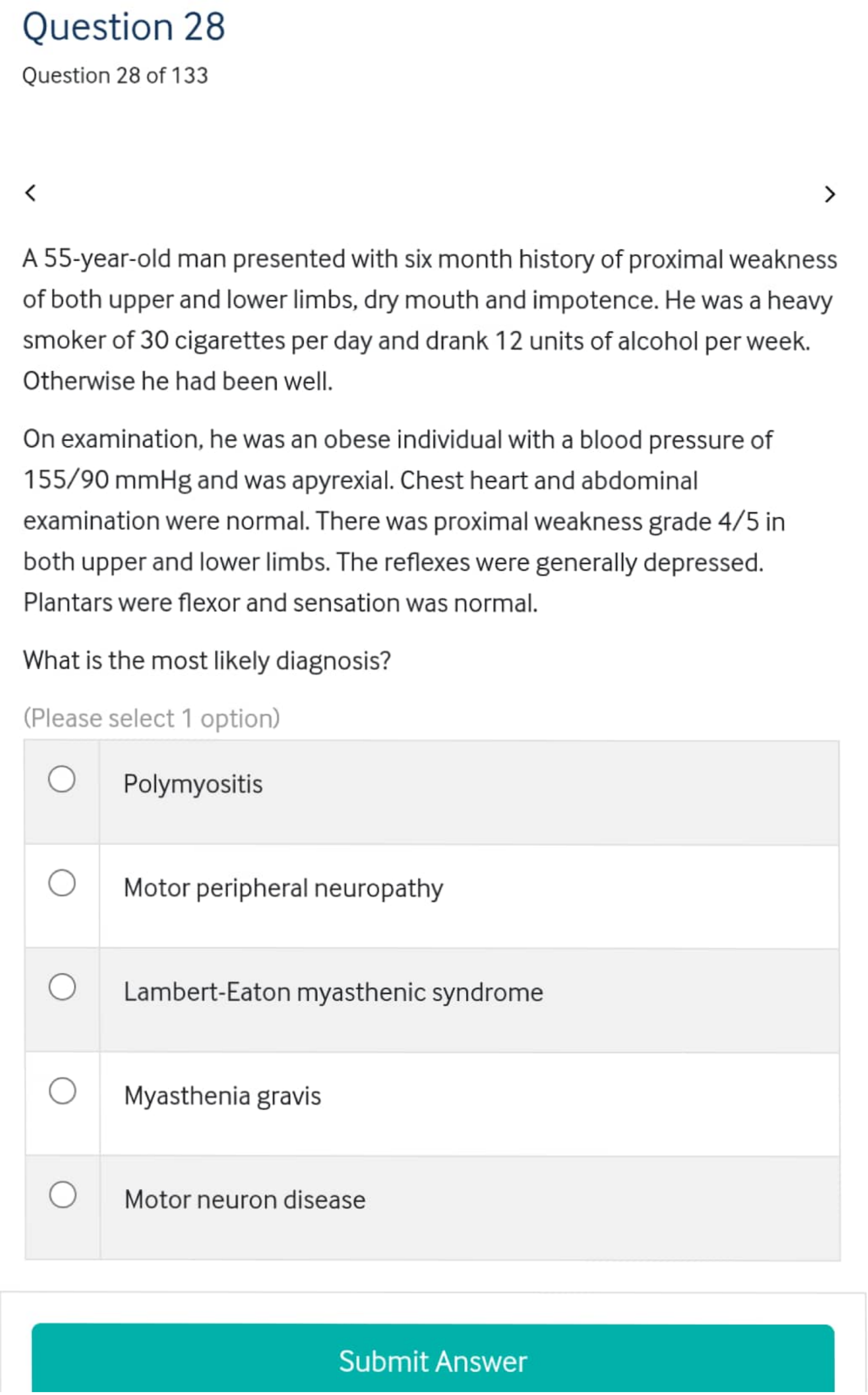


Question 28

Question 28 of 133

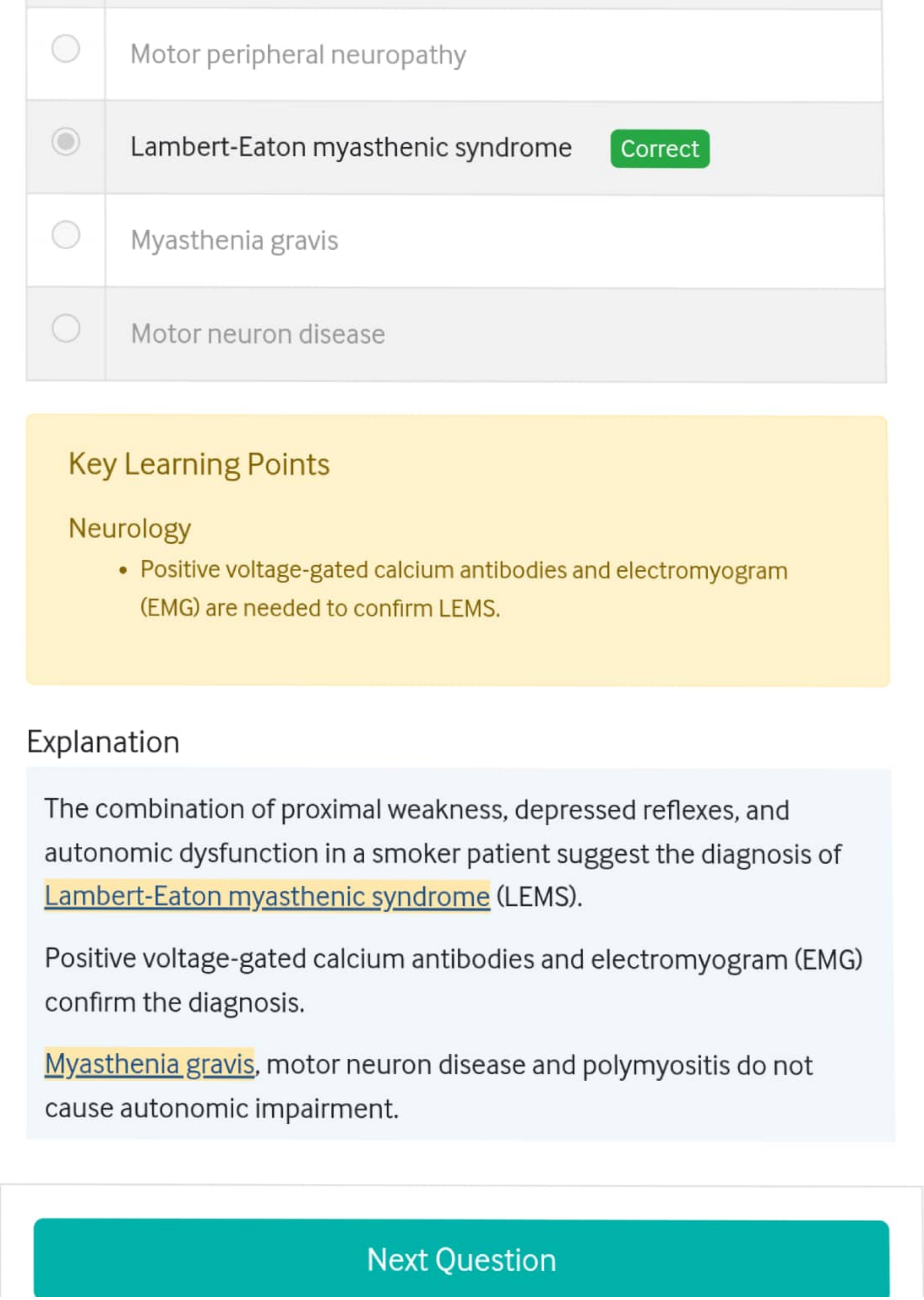
A 55-year-old man presented with six month history of proximal weakness of both upper and lower limbs, dry mouth and impotence. He was a heavy smoker of 30 cigarettes per day and drank 12 units of alcohol per week. Otherwise he had been well.

On examination, he was an obese individual with a blood pressure of 155/90 mmHg and was apyrexial. Chest heart and abdominal examination were normal. There was proximal weakness grade 4/5 in both upper and lower limbs. The reflexes were generally depressed. Plantars were flexor and sensation was normal.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Polymyositis
- ○ Motor peripheral neuropathy
- ○ Lambert-Eaton myasthenic syndrome
- ○ Myasthenia gravis
- ○ Motor neuron disease



○ Motor peripheral neuropathy

● Lambert-Eaton myasthenic syndrome

○ Myasthenia gravis

○ Motor neuron disease

Key Learning Points

Neurology

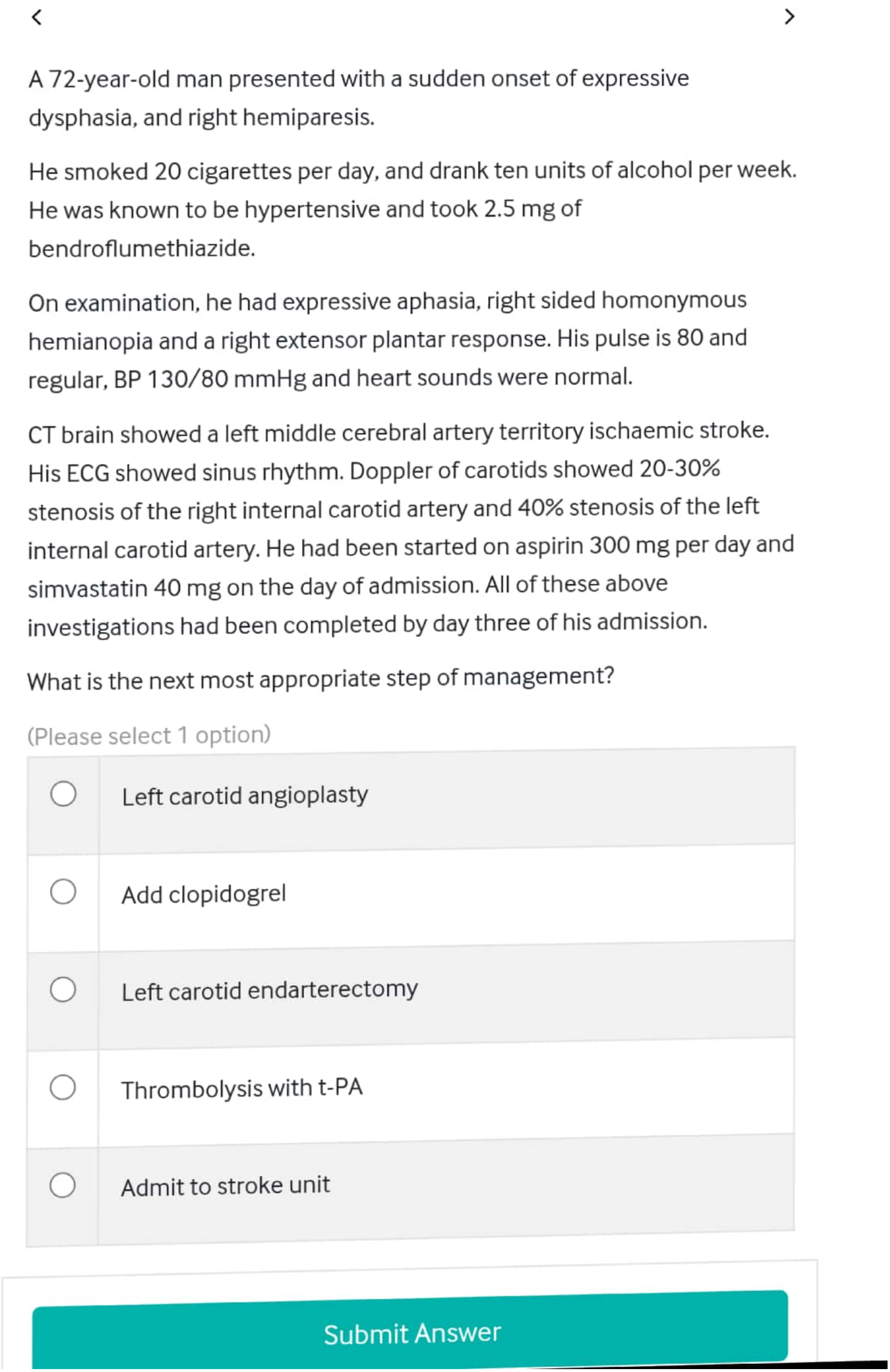
- Positive voltage-gated calcium antibodies and electromyogram (EMG) are needed to confirm LEMS.

Explanation

The combination of proximal weakness, depressed reflexes, and autonomic dysfunction in a smoker patient suggest the diagnosis of Lambert-Eaton myasthenic syndrome (LEMS).

Positive voltage-gated calcium antibodies and electromyogram (EMG) confirm the diagnosis.

Myasthenia gravis, motor neuron disease and polymyositis do not cause autonomic impairment.



A 72-year-old man presented with a sudden onset of expressive dysphasia, and right hemiparesis.

He smoked 20 cigarettes per day, and drank ten units of alcohol per week. He was known to be hypertensive and took 2.5 mg of bendroflumethiazide.

On examination, he had expressive aphasia, right sided homonymous hemianopia and a right extensor plantar response. His pulse is 80 and regular, BP 130/80 mmHg and heart sounds were normal.

CT brain showed a left middle cerebral artery territory ischaemic stroke. His ECG showed sinus rhythm. Doppler of carotids showed 20-30% stenosis of the right internal carotid artery and 40% stenosis of the left internal carotid artery. He had been started on aspirin 300 mg per day and simvastatin 40 mg on the day of admission. All of these above investigations had been completed by day three of his admission.

What is the next most appropriate step of management?

(Please select 1 option)

- ○ Left carotid angioplasty
- ○ Add clopidogrel
- ○ Left carotid endarterectomy
- ○ Thrombolysis with t-PA
- ○ Admit to stroke unit

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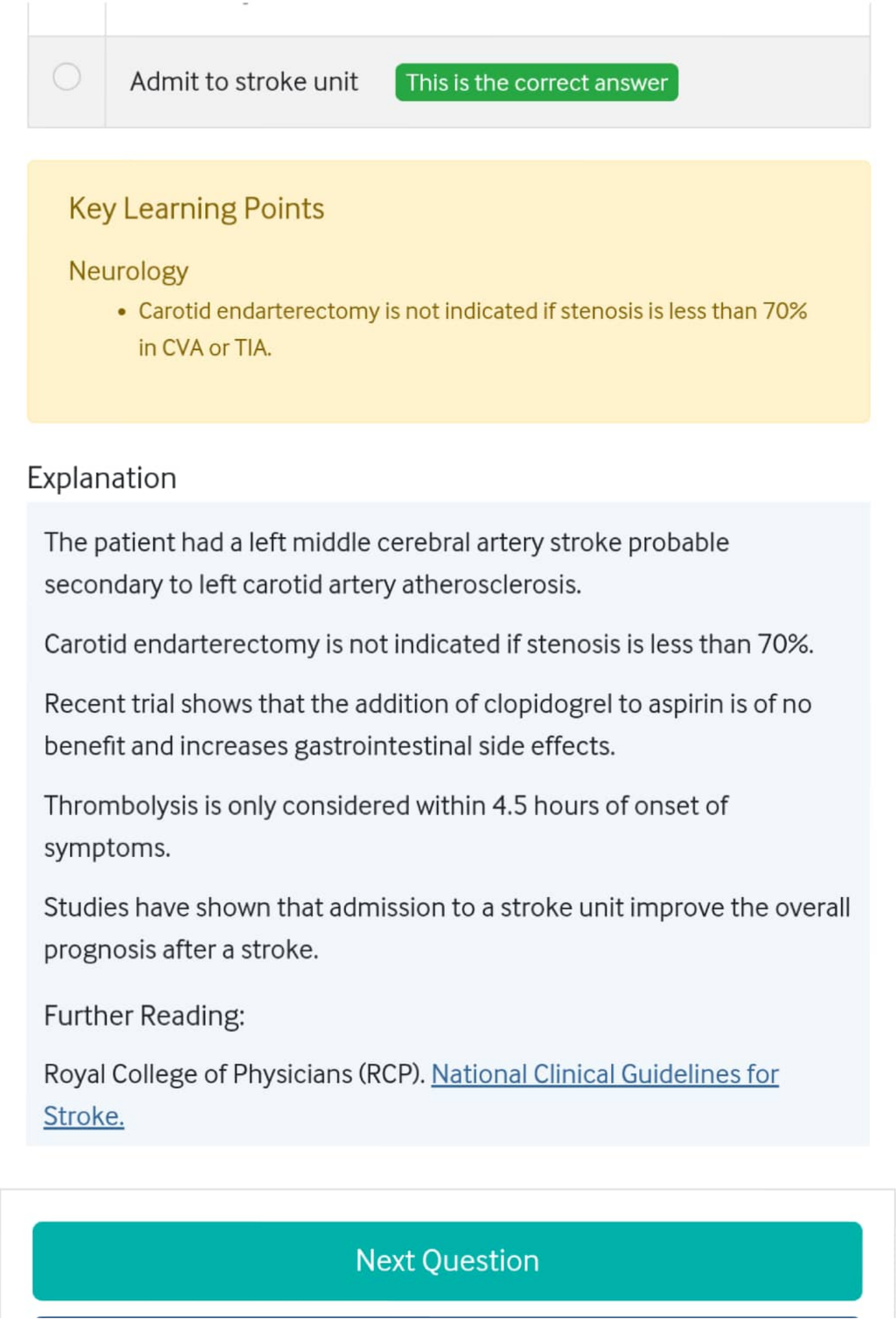

○ Admit to stroke unit **This is the correct answer**

Key Learning Points

Neurology

- Carotid endarterectomy is not indicated if stenosis is less than 70% in CVA or TIA.

Explanation

The patient had a left middle cerebral artery stroke probable secondary to left carotid artery atherosclerosis.

Carotid endarterectomy is not indicated if stenosis is less than 70%.

Recent trial shows that the addition of clopidogrel to aspirin is of no benefit and increases gastrointestinal side effects.

Thrombolysis is only considered within 4.5 hours of onset of symptoms.

Studies have shown that admission to a stroke unit improve the overall prognosis after a stroke.

Further Reading:

Royal College of Physicians (RCP). National Clinical Guidelines for Stroke.

A 65-year-old man was referred to a neurologist having developed weakness of his left foot.

He had noticed that he had been stubbing his toes whilst walking and had developed numbness over the entire foot which extended up the back of the thigh. He did not have any associated back pain and had not noticed any problems with bowel or bladder.

He had a history of longstanding diabetes and hypertension and took metformin 1 g/day, gliclazide 80 mg BD and ramipril 2.5 mg OD. He was a smoker of 10 cigarettes per day and drank four pints of beer at the weekend.

On examination, there was evidence of wasting of the left anterior compartments in the lower leg and mild wasting of the hamstrings. Tone was significantly reduced at the left ankle and the left foot had flaccid tone. There was marked weakness of foot dorsiflexion, plantar flexion, eversion and inversion with additional weakness of left hip extension and knee flexion. There was an absent ankle and plantar response on the left with all other reflexes intact. Sensory examination revealed reduced sensation to all modalities over the entire posterior left lower leg and the back of the left thigh.

Where is the likely lesion in this patient?

(Please select 1 option)

○ Sciatic nerve palsy

○ L5 radiculopathy

○ Peripheral neuropathy

○ Lumbar plexopathy

○ Common peroneal nerve palsy

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(Please select 1 option)

● Sciatic nerve palsy **Correct**

○ L5 radiculopathy

○ Peripheral neuropathy

○ Lumbar plexopathy

○ Common peroneal nerve palsy

Key Learning Points

Neurology

- The sciatic nerve (L4-S3) supplies both anterior and posterior compartment muscles and also the hamstrings.

Explanation

The clinical findings are consistent with left sciatic nerve palsy.

The sciatic nerve (L4-S3) supplies both anterior and posterior compartment muscles and also the hamstrings. The patient can develop weakness of the foot, knee flexion and hip extension.

Although sciatic nerve palsy is a rarity, it can occur following traumatic fracture of the hip, or as a result of an ill-placed intramuscular injection. Other causes include neurofibromas or nerve necrosis secondary to diabetes or infection.

A lumbar plexopathy would cause root pain. A common peroneal palsy would cause weakness of dorsiflexion and eversion only and a sensory disturbance affecting the outer lower leg and dorsum of the foot.

An L5 root lesion would cause weakness of dorsiflexion and inversion and a similar sensory disturbance to a common peroneal neuropathy. Reflexes in both these cases would be preserved.

A peripheral neuropathy would cause distal weakness, reflex change and sensory loss, which is not confined to a particular nerve or nerve root.

Next Question

A 23-year-old woman presents complaining of a 'droopy face' occurring over the last 24 hours.

Two weeks previously she had several days of headache and mild photophobia. The headache was of acute onset and moderately severe. Over the last week she has also noticed mild lower back discomfort and occasional tingling in the feet which she put down to the fact that she had 'pulled a muscle' when walking.Two months ago she had returned from a holiday in Thailand.

She takes the oral contraceptive pill. She smokes 20 cigarettes a day and consumes alcohol socially. She has a family history of migraine and her mother died of a 'brain haemorrhage'.

On examination, she looks well. Pulse 88 beats/min, BP 105/75 mmHg, temperature 37°C. On examination of her eyes, she has normal pupillary responses to light and a full range of eye movements. Fundoscopy is unremarkable. Corneal reflexes are present equally on both sides. She has difficulty closing her left eye and it rolls it upwards in the attempt. When the cheeks are puffed, the left side balloons more than the right.

The rest of the cranial nerve examination is unremarkable. Power is symmetrical in the upper and lower limbs and tone appears normal. She has absent reflexes throughout, even with reinforcement. Plantar responses are downgoing bilaterally. She is able to do finger to nose and heel to shin testing adequately. Sensory examination of light touch, pinprick and vibration sense are all normal.

What is the most likely diagnosis?

(Please select 1 option)

○ Cerebrovascular infarction

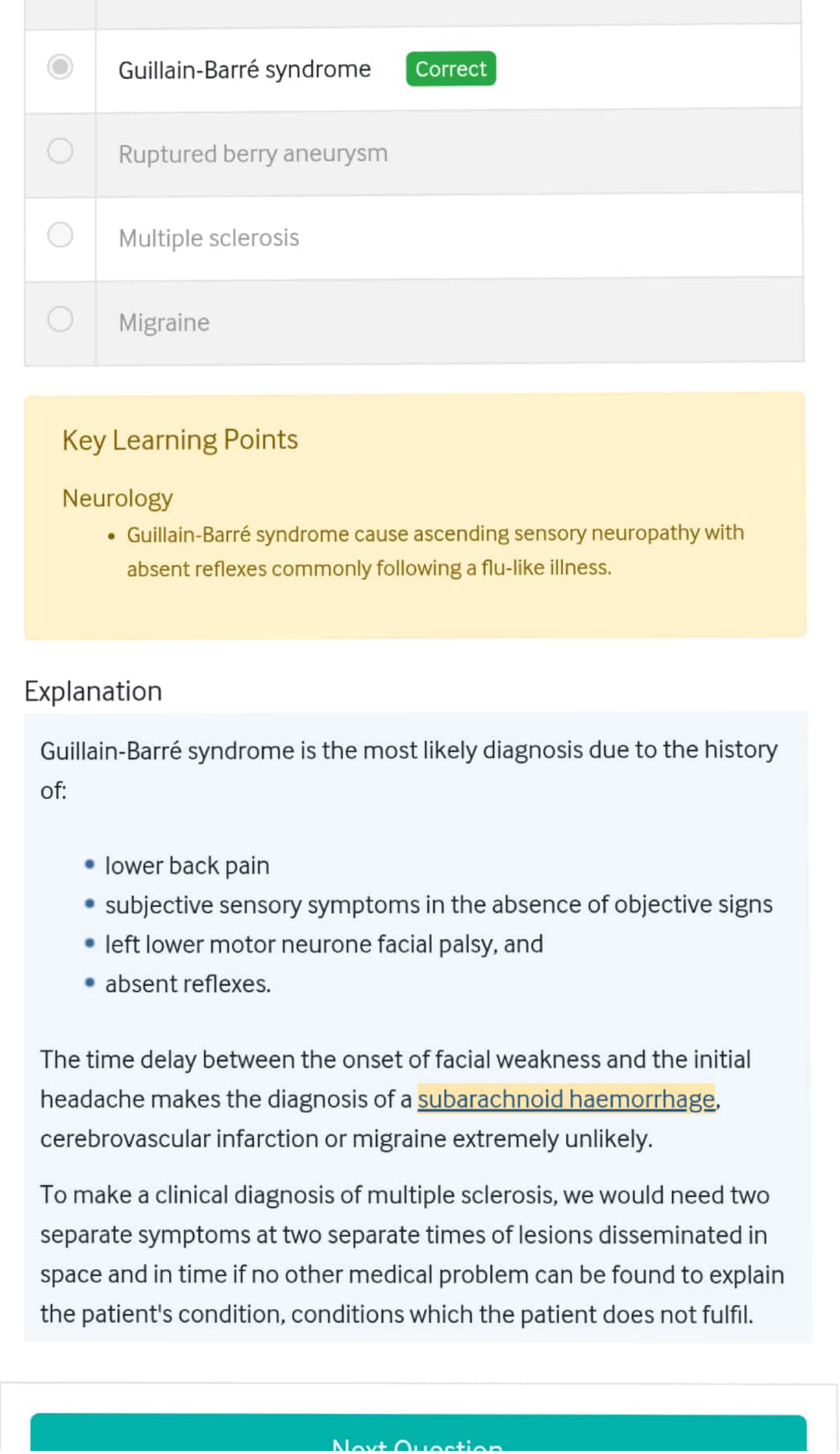
○ Guillain-Barré syndrome

○ Ruptured berry aneurysm

○ Multiple sclerosis

○ Migraine

Submit Answer



●	Guillain-Barré syndrome **Correct**
○	Ruptured berry aneurysm
○	Multiple sclerosis
○	Migraine

Key Learning Points

Neurology

- Guillain-Barré syndrome cause ascending sensory neuropathy with absent reflexes commonly following a flu-like illness.

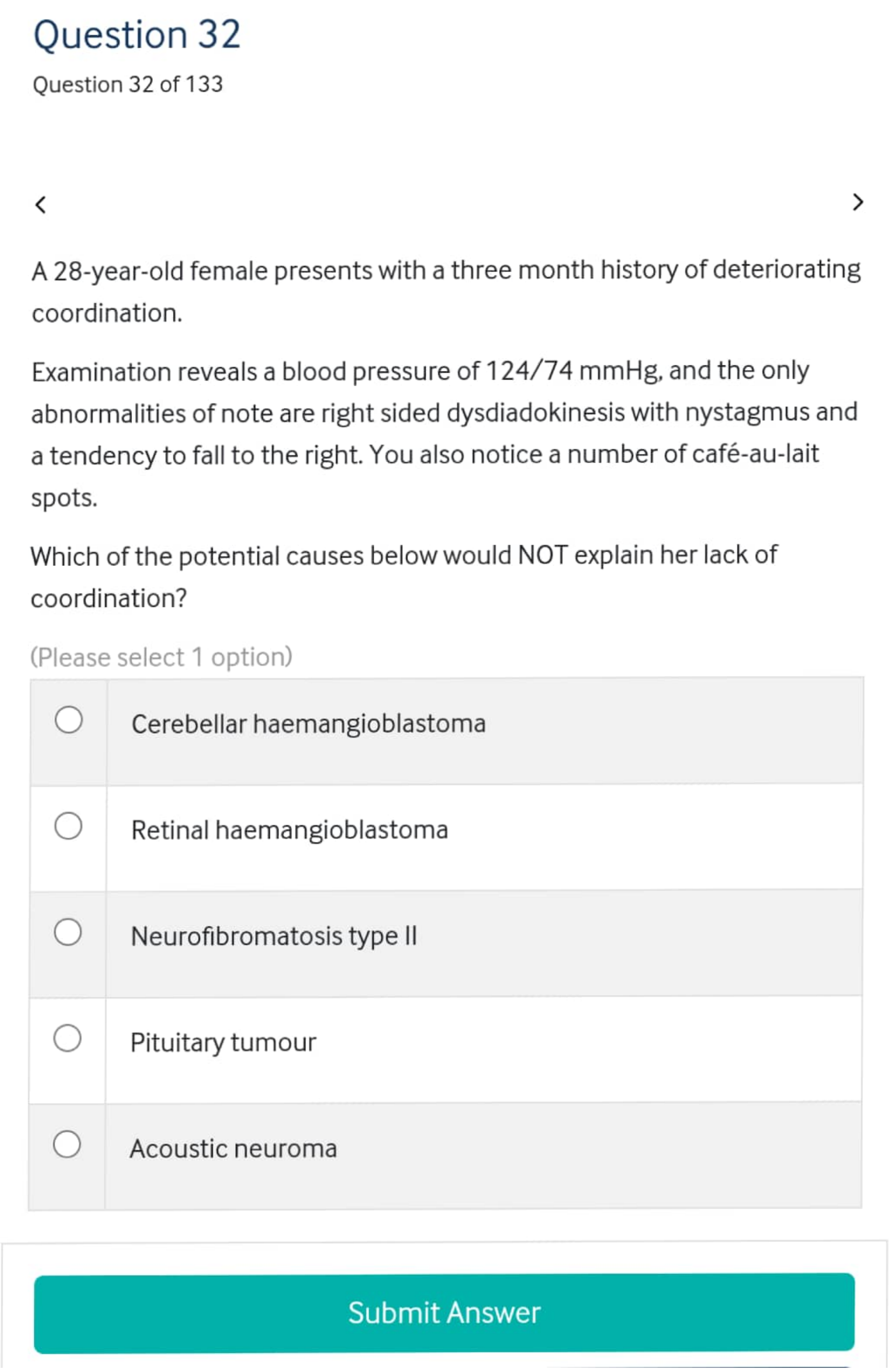
Explanation

Guillain-Barré syndrome is the most likely diagnosis due to the history of:

- lower back pain
- subjective sensory symptoms in the absence of objective signs
- left lower motor neurone facial palsy, and
- absent reflexes.

The time delay between the onset of facial weakness and the initial headache makes the diagnosis of a subarachnoid haemorrhage, cerebrovascular infarction or migraine extremely unlikely.

To make a clinical diagnosis of multiple sclerosis, we would need two separate symptoms at two separate times of lesions disseminated in space and in time if no other medical problem can be found to explain the patient's condition, conditions which the patient does not fulfil.



Question 32

Question 32 of 133

A 28-year-old female presents with a three month history of deteriorating coordination.

Examination reveals a blood pressure of 124/74 mmHg, and the only abnormalities of note are right sided dysdiadokinesis with nystagmus and a tendency to fall to the right. You also notice a number of café-au-lait spots.

Which of the potential causes below would NOT explain her lack of coordination?

(Please select 1 option)

- ○ Cerebellar haemangioblastoma
- ○ Retinal haemangioblastoma
- ○ Neurofibromatosis type II
- ○ Pituitary tumour
- ○ Acoustic neuroma

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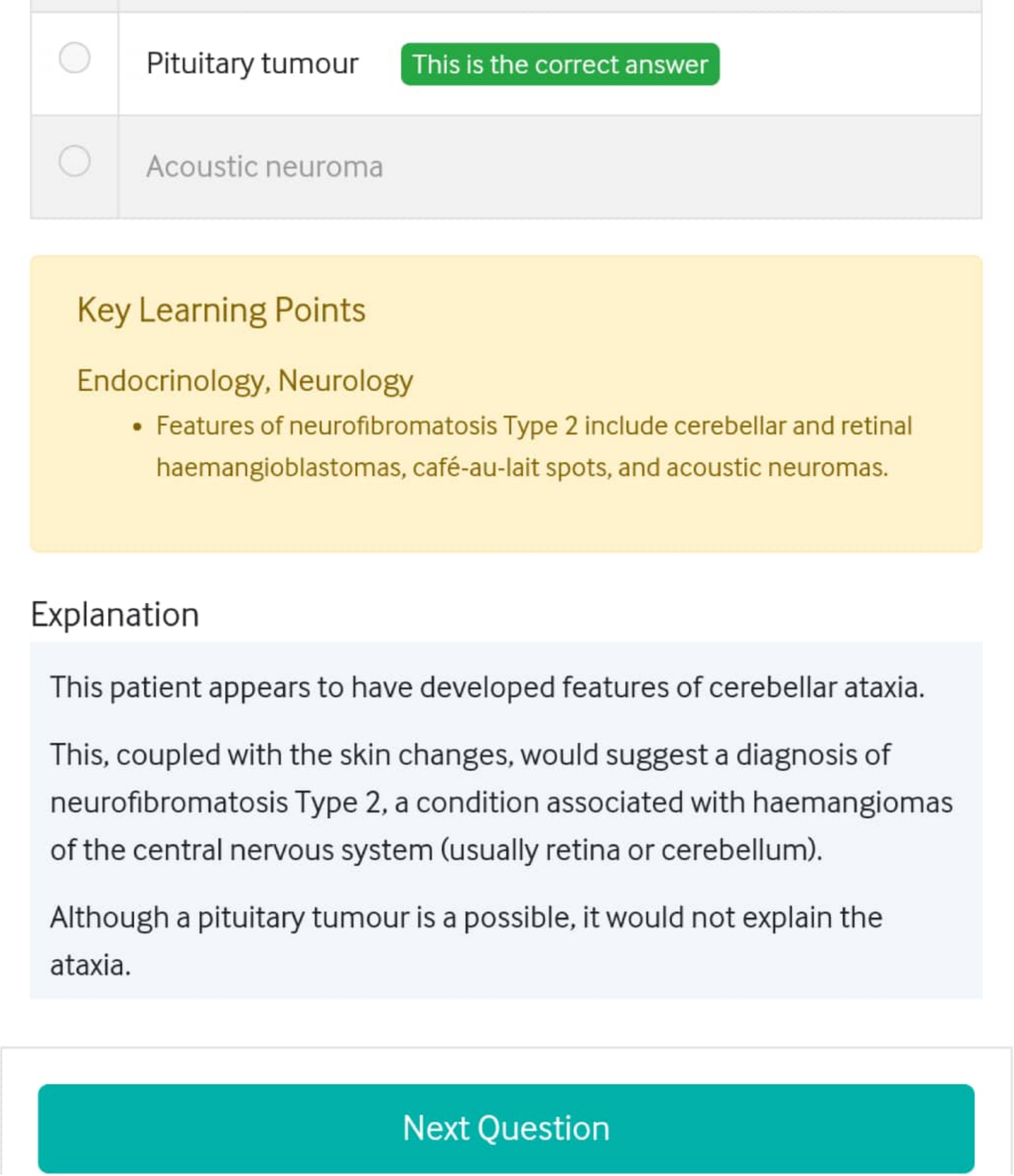

○ Pituitary tumour

○ Acoustic neuroma

Key Learning Points

Endocrinology, Neurology

- Features of neurofibromatosis Type 2 include cerebellar and retinal haemangioblastomas, café-au-lait spots, and acoustic neuromas.

Explanation

This patient appears to have developed features of cerebellar ataxia.

This, coupled with the skin changes, would suggest a diagnosis of neurofibromatosis Type 2, a condition associated with haemangiomas of the central nervous system (usually retina or cerebellum).

Although a pituitary tumour is a possible, it would not explain the ataxia.

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A 44-year-old woman attended the Emergency department having developed a severe sudden onset headache associated with nausea and vomiting.

In the last hour she had noticed blurring of vision and had also noticed intermittent uncontrollable twitching of her left hand. She was recovering from a bout of gastroenteritis and not been well in the last week.

She had a past medical history of psoriatic arthropathy. She did not take any regular medication, was a smoker of 35 cigarettes per day and drank approximately 10 units of alcohol per week.

On examination she appeared dehydrated, her blood pressure was 120/65 mmHg, pulse was 67 beats per minute and temperature was 36.5°C. Her jugular venous pressure was 6 cm, and auscultation of the chest revealed normal heart sounds. Chest examination appeared clear and there were no significant abnormalities in the abdominal examination.

Neurological examination revealed mild papilloedema with no other abnormalities within the cranial nerves. Peripheral nervous system examination revealed increased tone in the left arm and leg with brisk reflexes on the left and a left extensor plantar response. There was evidence of mild leg swelling but no obvious tenderness.

Investigations revealed:

Haemoglobin	112 g/L	(115-165)
White cell count	7.8 ×10^9/L	(4-11)
Platelets	100 ×10^9/L	(150-400)
Sodium	136 mmol/L	(137-144)
Potassium	3.3 mmol/L	(3.5-4.9)
Urea	10.6 mmol/L	(2.5-7.5)
Creatinine	110 μmol/L	(60-110)
Activated partial thromboplastin time	50 s	(30-40)

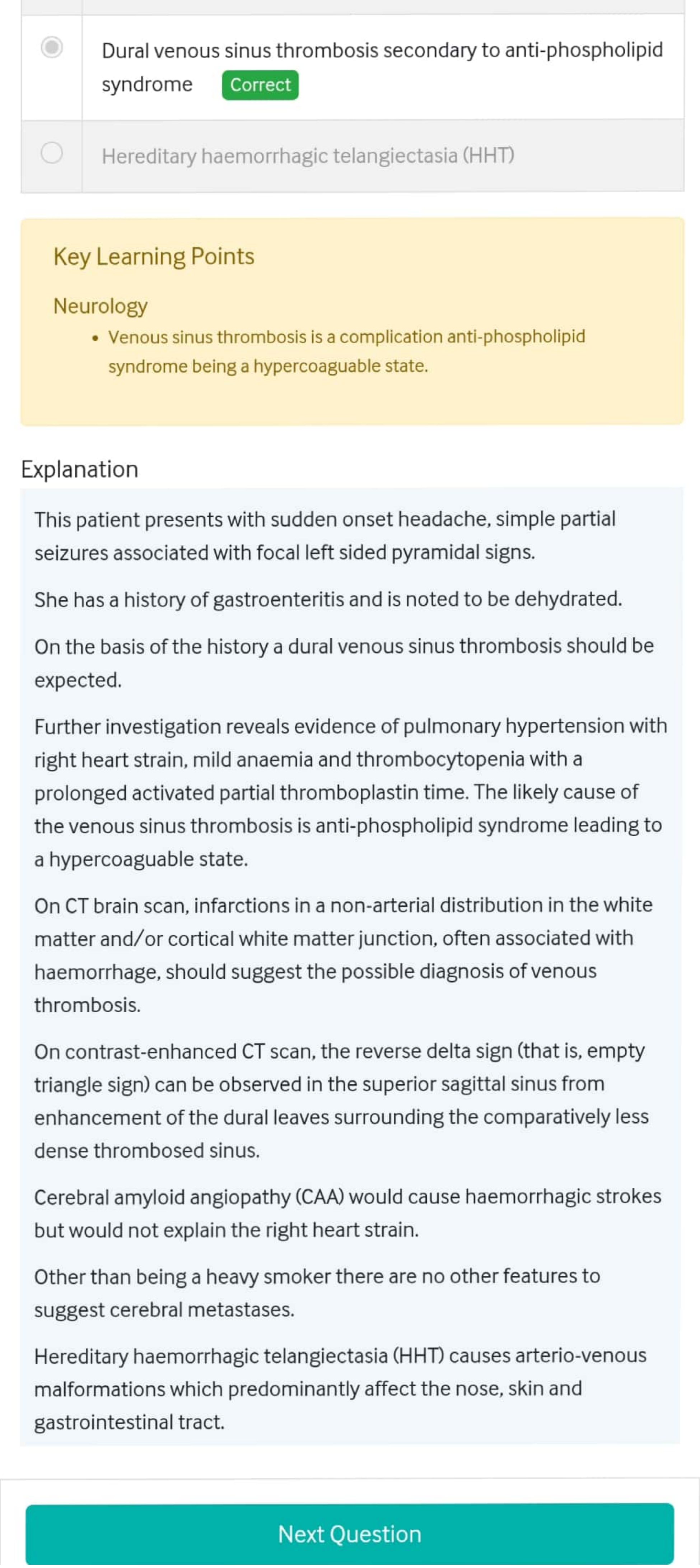
A CT brain scan (with contrast) showed an enhancing lesion within the right frontal cortical white matter junction associated with haemorrhage and cerebral oedema.

What is the most likely cause for the patient's neurological presentation?

(Please select 1 option)

- ○ Cerebral amyloid angiopathy (CAA)
- ○ Vasculitis associated with systemic lupus erythematosus (SLE)
- ○ Cerebral metastases secondary to lung carcinoma
- ○ Dural venous sinus thrombosis secondary to anti-phospholipid syndrome
- ○ Hereditary haemorrhagic telangiectasia (HHT)

Submit Answer



● Dural venous sinus thrombosis secondary to anti-phospholipid syndrome

○ Hereditary haemorrhagic telangiectasia (HHT)

Key Learning Points

Neurology

- Venous sinus thrombosis is a complication anti-phospholipid syndrome being a hypercoaguable state.

Explanation

This patient presents with sudden onset headache, simple partial seizures associated with focal left sided pyramidal signs.

She has a history of gastroenteritis and is noted to be dehydrated.

On the basis of the history a dural venous sinus thrombosis should be expected.

Further investigation reveals evidence of pulmonary hypertension with right heart strain, mild anaemia and thrombocytopenia with a prolonged activated partial thromboplastin time. The likely cause of the venous sinus thrombosis is anti-phospholipid syndrome leading to a hypercoaguable state.

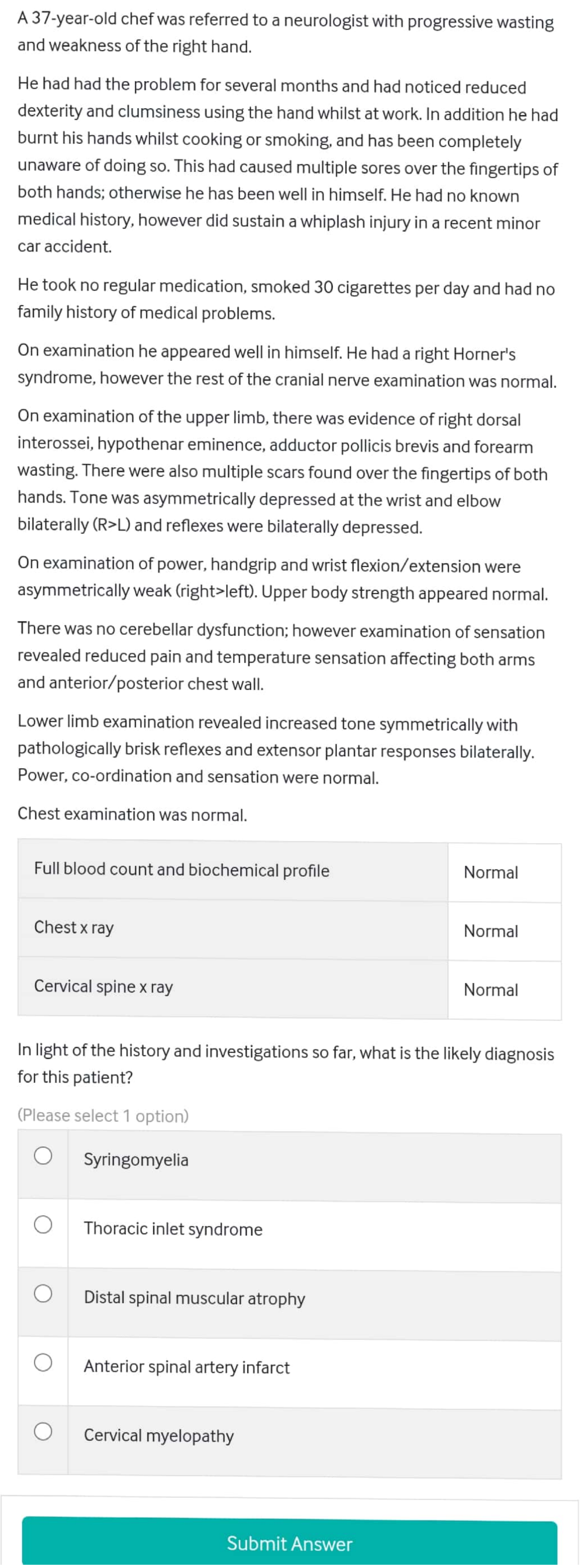
On CT brain scan, infarctions in a non-arterial distribution in the white matter and/or cortical white matter junction, often associated with haemorrhage, should suggest the possible diagnosis of venous thrombosis.

On contrast-enhanced CT scan, the reverse delta sign (that is, empty triangle sign) can be observed in the superior sagittal sinus from enhancement of the dural leaves surrounding the comparatively less dense thrombosed sinus.

Cerebral amyloid angiopathy (CAA) would cause haemorrhagic strokes but would not explain the right heart strain.

Other than being a heavy smoker there are no other features to suggest cerebral metastases.

Hereditary haemorrhagic telangiectasia (HHT) causes arterio-venous malformations which predominantly affect the nose, skin and gastrointestinal tract.



A 37-year-old chef was referred to a neurologist with progressive wasting and weakness of the right hand.

He had had the problem for several months and had noticed reduced dexterity and clumsiness using the hand whilst at work. In addition he had burnt his hands whilst cooking or smoking, and has been completely unaware of doing so. This had caused multiple sores over the fingertips of both hands; otherwise he has been well in himself. He had no known medical history, however did sustain a whiplash injury in a recent minor car accident.

He took no regular medication, smoked 30 cigarettes per day and had no family history of medical problems.

On examination he appeared well in himself. He had a right Horner's syndrome, however the rest of the cranial nerve examination was normal.

On examination of the upper limb, there was evidence of right dorsal interossei, hypothenar eminence, adductor pollicis brevis and forearm wasting. There were also multiple scars found over the fingertips of both hands. Tone was asymmetrically depressed at the wrist and elbow bilaterally (R>L) and reflexes were bilaterally depressed.

On examination of power, handgrip and wrist flexion/extension were asymmetrically weak (right>left). Upper body strength appeared normal.

There was no cerebellar dysfunction; however examination of sensation revealed reduced pain and temperature sensation affecting both arms and anterior/posterior chest wall.

Lower limb examination revealed increased tone symmetrically with pathologically brisk reflexes and extensor plantar responses bilaterally. Power, co-ordination and sensation were normal.

Chest examination was normal.

Full blood count and biochemical profile	Normal
Chest x ray	Normal
Cervical spine x ray	Normal

In light of the history and investigations so far, what is the likely diagnosis for this patient?

(Please select 1 option)

- ○ Syringomyelia
- ○ Thoracic inlet syndrome
- ○ Distal spinal muscular atrophy
- ○ Anterior spinal artery infarct
- ○ Cervical myelopathy

(Please select 1 option)

● Syringomyelia Correct

○ Thoracic inlet syndrome

○ Distal spinal muscular atrophy

○ Anterior spinal artery infarct

○ Cervical myelopathy

Key Learning Points

Neurology

- Consider syringomyelia in context of progressive pain and weakness in back, shoulders, arms or legs with dissociated sensory disturbances and bowel/bladder dysfunction.

Explanation

This patient presents with wasting and weakness of the hands, dissociated sensory disturbance with painless burns in the upper limb, and pyramidal signs in the lower limbs.

These findings would all be in keeping with a diagnosis of syringomyelia affecting the cervical cord.

There is sympathetic involvement as the patient has a right Horner's syndrome.

The syrinx does not extend to involve the brainstem, as there is no cranial nerve dysfunction involving cranial nerves V, VII, IX and X.

A magnetic resonance imaging (MRI) scan is required to visualise the lesion and neurosurgical opinion. Treatment options include syringoperitoneal shunt or direct drainage of the syrinx into the subarachnoid space.

Anterior spinal artery infarct would have resulted in an acute anterior cord syndrome with pyramidal signs and dissociated sensory disturbance affecting the legs and lower motor neurone (LMN) signs at the level of the infarct (T4-T6 watershed).

Distal spinal muscular atrophy can resemble amyotrophic lateral sclerosis but is limited to pure lower motor neurone degeneration. This condition would not explain the prominent sensory disturbance and the Horner's syndrome.

Thoracic inlet syndrome involving the lower brachial plexus would cause a C8/T1 sensory and motor dysfunction and possibly absent brachial or radial pulsation.

Cervical myelopathy usually affects C6, C7 roots and is unlikely to account for the gross wasting of the right hand. Joint position and vibration sense are often lost in the legs and there is often very little sensory loss of the hands.

Next Question

A 53-year-old woman presented to her GP with a three month history of lethargy associated with weakness of her shoulders and difficulty swallowing. On several occasions she had experienced nasal regurgitation. In the last few weeks she had noticed marked difficulty combing her hair, or placing objects on a high shelf. She had also developed an aching of her legs, particularly her thighs. She denied any diplopia or visual disturbances.

She had a medical history of rheumatic fever as a child, but had no other conditions and took no regular medication. She was a smoker of 30 cigarettes per day and did not drink any alcohol.

On examination she was alert and orientated. Her blood pressure was 134/78 mmHg, pulse was 67/min and regular and oxygen saturation was 96% on room air. On examination of the chest there was bilateral expiratory wheeze with no other findings. Cranial nerve examination revealed normal ocular movements, facial movements and tongue movements. There was, however, some weakness of neck flexion and extension. On upper limb examination there was marked proximal weakness, but normal tone and sensation. On lower limb examination there was weakness of hip flexion and extension, with normal tone, distal power and sensation.

Investigations revealed:

Haemoglobin	102 g/L	(130-180)
Mean cell volume	86 fL	(80-96)
White cell count	11.0×10^9/L	(4-11)
Platelets	456×10^9/L	(150-400)
ESR (Westergren)	150 mm/1st hour	(0-15)
Serum sodium	138 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	4.5 mmol/L	(2.5-7.5)
Serum creatinine	97 µmol/L	(60-110)
Serum creatine kinase	3000 U/L	(24-170)
Serum albumin	41 g/L	(37-49)
Serum bilirubin	19 µmol/L	(1-22)
Serum AST	24 U/L	(1-31)
Serum alkaline phosphatase	47 U/L	(45-105)
Seum gamma-GT	32 U/L	(4-35)

The chest x ray showed a left hilar mass.

An electromyogram (EMG) showed spontaneous fibrillations and short polyphasic motor units.

What is the likely diagnosis in this patient?

(Please select 1 option)

○ Myasthenia gravis

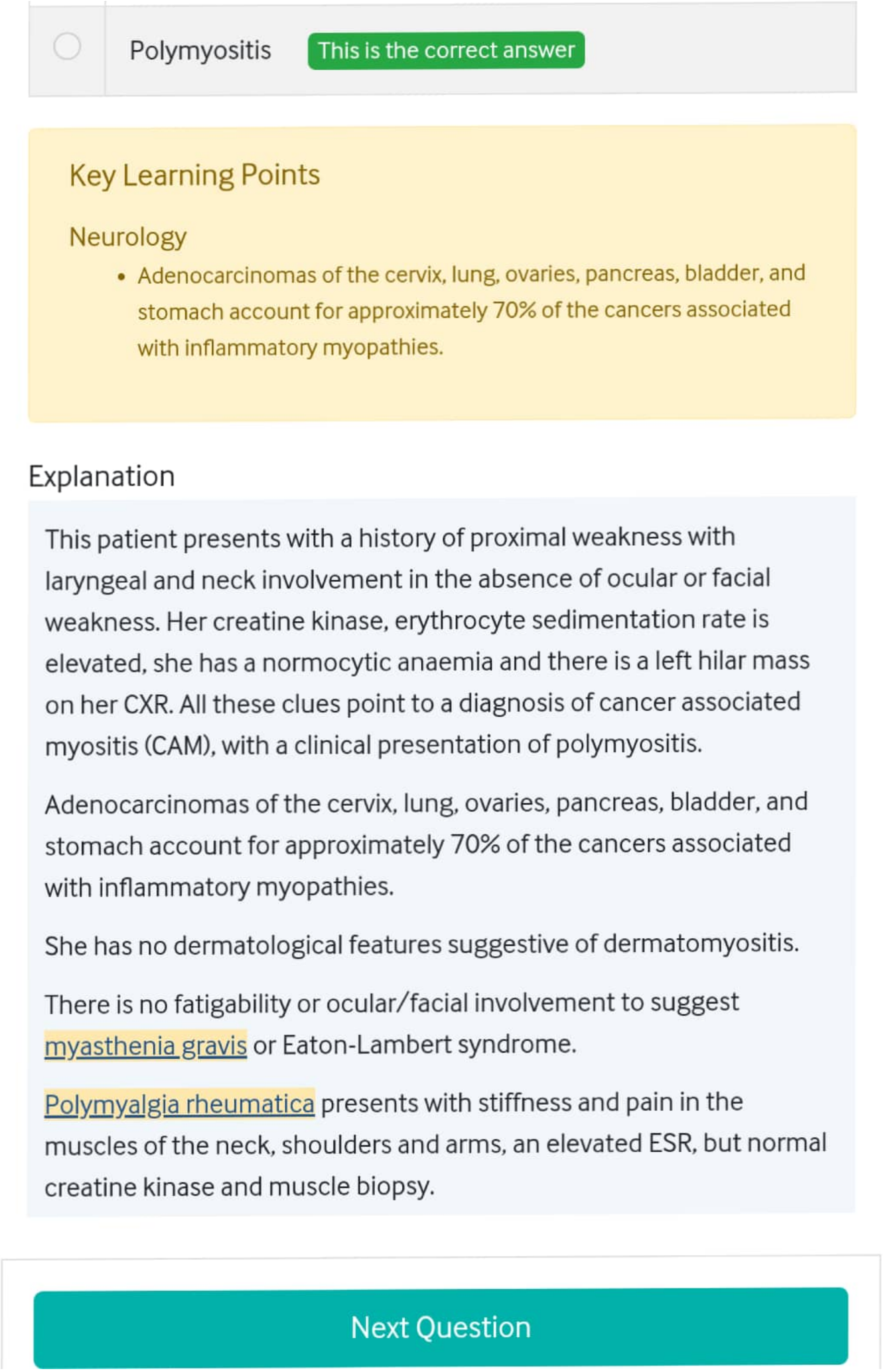
○ Dermatomyositis

○ Eaton-Lambert syndrome

○ Polymyalgia rheumatica

○ Polymyositis

Submit Answer



○ Polymyositis **This is the correct answer**

Key Learning Points

Neurology

- Adenocarcinomas of the cervix, lung, ovaries, pancreas, bladder, and stomach account for approximately 70% of the cancers associated with inflammatory myopathies.

Explanation

This patient presents with a history of proximal weakness with laryngeal and neck involvement in the absence of ocular or facial weakness. Her creatine kinase, erythrocyte sedimentation rate is elevated, she has a normocytic anaemia and there is a left hilar mass on her CXR. All these clues point to a diagnosis of cancer associated myositis (CAM), with a clinical presentation of polymyositis.

Adenocarcinomas of the cervix, lung, ovaries, pancreas, bladder, and stomach account for approximately 70% of the cancers associated with inflammatory myopathies.

She has no dermatological features suggestive of dermatomyositis.

There is no fatigability or ocular/facial involvement to suggest myasthenia gravis or Eaton-Lambert syndrome.

Polymyalgia rheumatica presents with stiffness and pain in the muscles of the neck, shoulders and arms, an elevated ESR, but normal creatine kinase and muscle biopsy.

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A 70-year-old man with Parkinson's disease is seen with his wife. He was diagnosed many years ago and has been taking a levodopa preparation (co-beneldopa) four times a day. He is on the maximum daily dose.

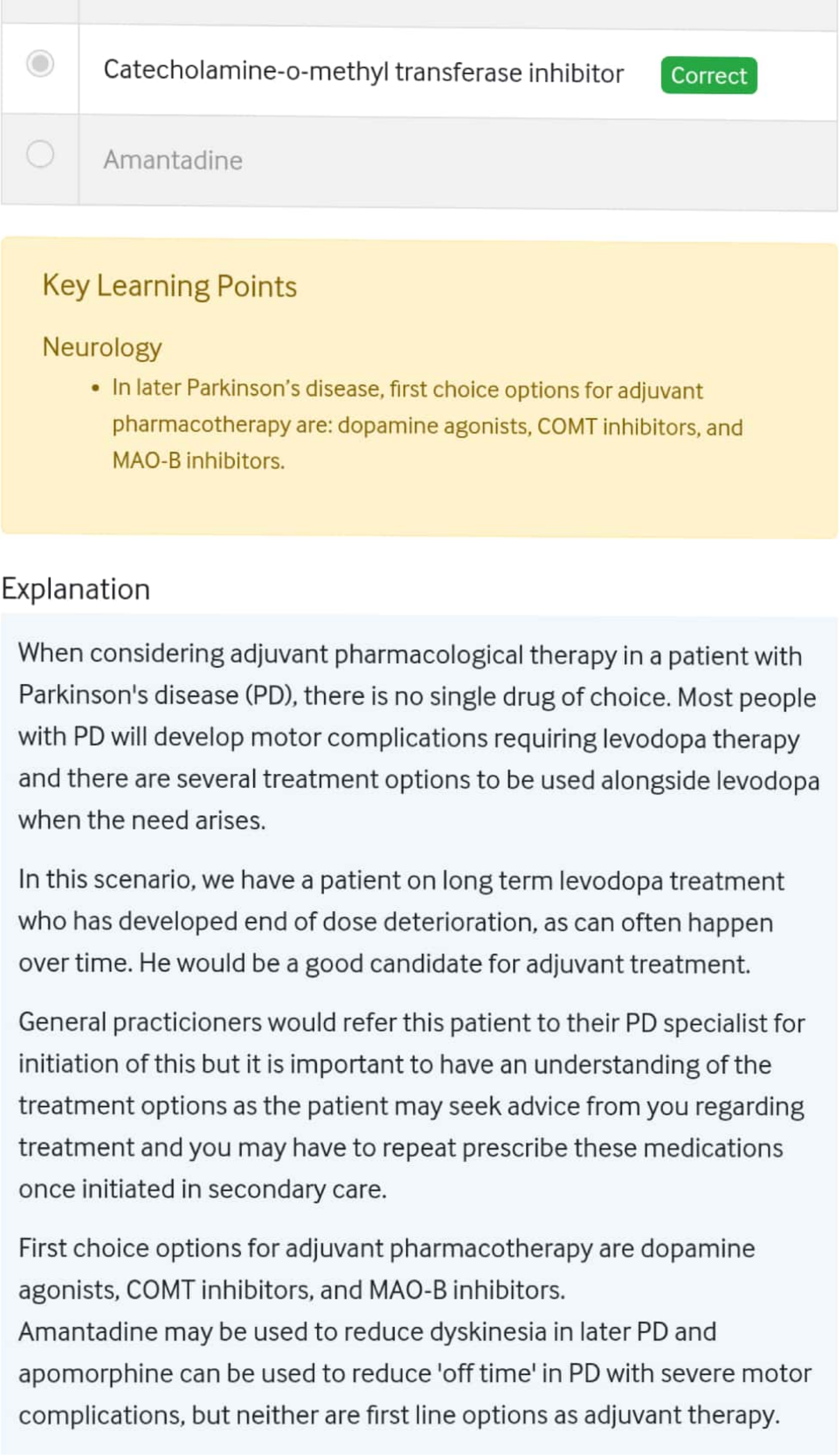
He has noticed that his symptoms seem to be becoming progressively more troublesome and are returning within a couple of hours of taking his medication.

According to NICE guidelines, which of the following would be the most appropriate additional pharmacotherapy to consider at this point in this patient?

(Please select 1 option)

- ○ Beta blocker
- ○ Anticholinergic
- ○ Apomorphine
- ○ Catecholamine-o-methyl transferase inhibitor
- ○ Amantadine

Submit Answer



● Catecholamine-o-methyl transferase inhibitor **Correct**

○ Amantadine

Key Learning Points

Neurology

- In later Parkinson's disease, first choice options for adjuvant pharmacotherapy are: dopamine agonists, COMT inhibitors, and MAO-B inhibitors.

Explanation

When considering adjuvant pharmacological therapy in a patient with Parkinson's disease (PD), there is no single drug of choice. Most people with PD will develop motor complications requiring levodopa therapy and there are several treatment options to be used alongside levodopa when the need arises.

In this scenario, we have a patient on long term levodopa treatment who has developed end of dose deterioration, as can often happen over time. He would be a good candidate for adjuvant treatment.

General practicioners would refer this patient to their PD specialist for initiation of this but it is important to have an understanding of the treatment options as the patient may seek advice from you regarding treatment and you may have to repeat prescribe these medications once initiated in secondary care.

First choice options for adjuvant pharmacotherapy are dopamine agonists, COMT inhibitors, and MAO-B inhibitors.
Amantadine may be used to reduce dyskinesia in later PD and apomorphine can be used to reduce 'off time' in PD with severe motor complications, but neither are first line options as adjuvant therapy.

A 53-year-old Asian woman with known HT, IHD, type 2 diabetes mellitus and colon cancer resected two years ago, presents to the Emergency department with a one week history of cough and shortness of breath.

She suffered from chronic mild right ear ache with some discharge and mild right sided headache with no reported neck stiffness or photophobia. Occasionally, she has been choking when drinking and since then her voice became hoarse.

On examination she was afebrile and had normal vital signs. There were scarce crepitations on the right base of lung. She had normal GCS and pupil response.

Cranial nerve examination revealed deviation of uvula to the left and her left palatolingual fold was positioned higher than the right. On protrusion, the tip of the tongue was deviated to the right. Limb examination was normal with negative Babinski's sign bilaterally.

Blood results showed:

Sodium	137 mmol/L	(137-144)
Potassium	4.0 mmol/L	(3.5-4.9)
Creatinine	83 µmol/L	(60-110)
Urea	6.2 mmol/L	(2.5-7.5)
Albumin	36 g/L	(37-49)
Total bilirubin	14 µmol/L	(1-22)
Alk. phosphatase	77 IU/L	(30-110)
ALT	14 IU/L	(5-40)
ESR	67 mm/hr	(0-10)
CRP	8 mg/L	(<5)
Hb	131 g/L	(130-180)
WBC	10.2 $\times 10^9$/L	(4-11)
Platelets	200 $\times 10^9$/L	(150-400)
Neutrophils	6.0 $\times 10^9$/L	(1.5-7.0)

What is the most likely diagnosis?

(Please select 1 option)

○	Leptomeningeal carcinomatosis
○	*Listeria monocytogenes* meningitis
○	Tuberculous meningitis
○	Skull base osteomyelitis
○	Leptomeningeal sarcoidosis

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● Skull base osteomyelitis **Correct**

○ Leptomeningeal sarcoidosis

Key Learning Points

Neurology

- Patients with multiple cranial nerve palsies should be considered for investigation of skull base osteomyelitis.

Explanation

This is a case of skull base osteomyelitis and there are clinical clues to it - a diabetic patient with otitis externa signs complicated by unilateral headache and lower motor neurone (LMN) signs, cranial nerve involvement of jugular foramen content and XIIth CN on the affected side with subsequent bulbar palsy presentation and dysphagia.

This is a rare but potentially life threatening condition affecting people with compromised immunity. Typically, *Pseudomonas aeruginosa* is the causative pathogen. Less common pathogens are *Proteus mirabilis*, *Staphylococcus aureus*, *Staphylococcus epidermidis*.

Usually osteomyelitis of the skull is preceded by a local infection, for example:

- Sinusitis extending to the sphenoid sinuses and involving frontal bone may have serious complications such as cavernous sinus thrombosis
- Mastoid cell infection and occipital bone osteomyelitis
- Necrotising otitis externa, complicated by petrous bone osteomyelitis with cranial nerve involvement (most common site of skull base osteomyelitis).

The clinical scenario depends on the affected part of the skull base in its most common form, that is, petrous bone involvement. Patients suffer from chronic otitis externa with otalgia and otorrhoea, which, if untreated, progress and cause unilateral headache, cranial nerve palsies, most commonly IX, X, XI (jugular foramen content) and include also XII nerve form, Villaret's syndrome.

The usual biochemical picture is raised erythrocyte sedimentation rate (ESR) and normal white cell count (WCC) and C reactive protein (CRP).

The typical imaging finding are signs of bone destruction especially clivus, shown as hypointensity of bone marrow in the clivus and preclival soft tissue infiltration on MRI T1 weighted images.

Diagnosis is confirmed by fine needle aspiration (FNA) of tissue and cultures.

Treatment is with antibiotics.[1]

In this scenario clinical suspicion should be raised to base of skull pathology, demonstrated by cranial nerve involvement. All of the above conditions neurologically may present similarly, but differential judgment should take facts from history and examination.

The infiltration of the leptomeninges by malignant cells is not an uncommon complication of cancer and is called carcinomatous meningitis or leptomeningeal carcinomatosis.[4]

The most common associated malignancies are breast and lung cancer, melanoma, lymphomas and leukaemias.[5,6]

In this scenario, there is no suggestion in the history of recurrence of the malignancy and colon cancer is not involved in the above group of malignancies causing carcinomatous infiltration.

Sarcoidosis is a multisystemic idiopathic granulomatous disease. The usual presentation is of young adults aged 20-40 years with chronic non-productive cough, gradually worsening dyspnoea and extrapulmonary presentation involving eye, lymph nodes, skin and joints. Around 5% of patients may have involvement of the central nervous system (CNS) as leptomeningeal sarcoidosis, cranial neuropathy and hydrocephalus.[2]

Listeria monocytogenes meningitis is clinically indistinguishable from other forms of bacterial meningitis but is a life threatening condition in elderly immune compromised patients and gentamicin and ampicillin should be considered in treatment. This is not the right answer here as the patient is under 70, is not severely immune compromised and does not present with meningism.[3]

Although some of the risk factors for TB are present in this scenario (diabetes mellitus, Asian ethnicity) and involvement of basal meninges in tuberculous meningitis is common, this is not the right answer because of the lack of other common clinical symptoms and signs coexistent in extrapulmonary Tb such as the involvement of lymph nodes, urinary tract, bones and constitutional signs.

A 32-year-old singing teacher presented to the Emergency department with a four day history of worsening weakness affecting both legs.

She had noticed marked difficulty walking downstairs and rising up out of a chair. She complained of severe lower back pain although denied any history of trauma and had not noticed any problems with her bowel or bladder. In the last 24 hours she had also developed some facial weakness and had been unable to sing. She denied any sensory disturbance. She did mention she had cut her finger on a rose trellis whilst out gardening in the last week otherwise she had felt well in herself.

She had a past medical history of psoriasis and hypothyroidism and took regular thyroxine. She was a non-smoker and did not drink any alcohol.

On examination her blood pressure was 135/86 mmHg, pulse was 87/min and regular and temperature was 36.7°C. Heart sounds were normal and chest was clear. On cranial nerve examination there was some mild bilateral facial weakness and ptosis. Pupillary responses were intact and the rest of the cranial nerves were normal.

Neck flexion and extension was intact. Upper limb examination revealed some mild weakness of shoulder abduction and adduction, which was non-fatigable with intact distal power. Tone and sensation were both intact however reflexes were depressed. Examination of the lower limb revealed marked weakness throughout (2/5), reduced tone and absent knee, ankle reflexes. Plantar responses were mute. Sensory examination was normal.

Investigations revealed:

Haemoglobin	147 g/L	(130-180)
White cell count	6.4×10^9/L	(4-11)
Platelets	199×10^9/L	(150-400)
ESR (Westergren)	18 mm/1st hour	(0-15)
Serum sodium	136 mmol/L	(137-144)
Serum potassium	4.5 mmol/L	(3.5-4.9)
Serum urea	4.2 mmol/L	(2.5-7.5)
Serum creatinine	89 μmol/L	(60-110)
Serum creatine kinase	102 U/L	(24-170)

A lumbar puncture was performed and yielded the following data:

Opening pressure	13 cmH_2O	(5-18)
CSF protein	0.95 g/L	(0.15-0.45)
CSF white cell count	4 cells per ml	(<5)
CSF red cell count	2 cells per ml	(<5)

What is the likely diagnosis in this patient?

(Please select 1 option)

○ Guillain-Barré syndrome

○ Myasthenia gravis

○ Viral myositis

○ Botulism

○ Vasculitic neuropathy

Submit Answer

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● Guillain-Barré syndrome **Correct**

○ Myasthenia gravis

○ Viral myositis

○ Botulism

○ Vasculitic neuropathy

Key Learning Points

Neurology

- GBS may present as a variant and affect purely motor neurons.

Explanation

This patient presents with acute symmetrical proximal weakness and facial weakness, with back pain and depressed reflexes in both upper limb and lower limb. There is an elevated cerebrospinal fluid (CSF) protein in absence of a leucocytosis.

The diagnosis is a pure motor variant form of Guillain-Barré syndrome.

The history and clinical findings are not in keeping with a diagnosis of botulism although this should always be a differential diagnosis of individuals presenting with rapidly progressive weakness, particularly if there is a history of intravenous drug abuse.

Other signs in keeping with botulism would include autonomic features particularly:

- vomiting
- abdominal pain
- dry mouth
- sluggish pupillary responses, and
- a descending flaccid paralysis affecting cranial nerves.

Depressed reflexes and elevated CSF protein would not be features in keeping with a diagnosis of myasthenia gravis.

Vasculitic neuropathy presents as a painful distal neuropathy or a mono-neuritis multiplex. In absence of raised inflammatory markers, and other organ involvement the diagnosis is unlikely.

A normal creatine kinase and depressed reflexes would go against a viral myositis.

Next Question

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A 50-year-old woman presented to her GP with a four month history of progressive distal sensory loss and weakness of both legs and arms. The weakness and numbness had extended to the elbows and knees.

On examination, cranial nerves and fundoscopy were normal. Examination of the upper limb revealed bilaterally reduced tone and 3/5 power.

Lower limb examination revealed some mild weakness of hip flexion and extension with marked weakness of dorsiflexion and plantarflexion. Both knee and ankle jerks were absent and both plantar responses were mute. There was absent sensation to all modalities affecting both feet extending to the knees.

A lumbar puncture was performed and yielded the following data:

Opening pressure	14 cm H2O	(5-18)
CSF protein	0.75 g/L	(0.15-0.45)
CSF white cell count	10 cells per ml	(<5 cells)
CSF white cell differential	90% lymphocytes	-
CSF red cell count	2 cells per ml	(<5 cells)

Nerve conduction studies showed multifocal motor and sensory conduction block with prolonged distal latencies.

What is the likely diagnosis in this patient?

(Please select 1 option)

○ Guillain-Barré syndrome

○ Multifocal motor neuropathy

○ Chronic inflammatory demyelinating neuropathy (CIDP)

○ Hereditary motor and sensory neuropathy (HMSN)

○ Cervical spondylosis

Submit Answer

○ Chronic inflammatory demyelinating neuropathy (CIDP)
This is the correct answer

○ Hereditary motor and sensory neuropathy (HMSN)

○ Cervical spondylosis

Key Learning Points

Neurology

- CIDP shows progressive weakness and sensory function in the upper and lower limbs.

Explanation

The history is compatible with a subacute sensory and motor peripheral neuropathy.

Causes of such conditions include inflammatory neuropathies such as chronic inflammatory demyelinating polyneuropathy (CIDP) and paraproteinaemic neuropathies.

CIDP is characterised by progressive weakness and impaired sensory function in the upper and lower limbs. The cause of the demyelination is not understood, but it is more common in young adults and in men. It presents with abnormal sensation (which typically begins distally), weakness of the limbs, areflexia and fatigue.

Treatment for CIDP includes corticosteroids, plasmapheresis and intravenous immunoglobulin. Physiotherapy is an effective adjunct. The course varies widely, and patients may be left with residual neurology or suffer a number of relapses.

Guillain-Barré syndrome (GBS) is an acute post-infectious neuropathy, thought possibly to be a post-infectious phenomenon, which reaches its peak in severity within six weeks. CIDP is closely linked to GBS, and is thought by some to be its chronic counterpart. Both CIDP and GBS can affect motor and sensory nerves, and it is the four month history which distinguishes the two here.

Cervical spondylosis would cause upper motor neurone signs such as hyperreflexia, extensor plantar response and possibly a sensory level.

HMSN is normally a very chronic neuropathy developing over many years and usually with a family history of the condition.

Multifocal motor neuropathy is a treatable neuropathy affecting motor conduction only. It is associated with antibodies to a ganglioside component of peripheral myelin.

Further Reading:

National Institute of Neurological Disorders and Stroke. Chronic Inflammatory Demyelinating Polyneuropathy (CIDP).

Next Question

A 48-year-old nurse was admitted to the Emergency department with a one week history of memory dysfunction and confusion.

In the last three days she had developed some numbness and weakness affecting her right arm and leg. She denied any headache or rash but had had flu-like illness prior to the onset of symptoms. She had a past medical history of eczema and had recently had a number of pre-holiday vaccinations for a trip to India. There was no family history of medical problems.

On examination she appeared flushed and confused. Her Glasgow coma scale was 14/15. Her blood pressure was 135/78 mmHg. Pulse was 78/min and regular and temperature was 37.8°C. Pupils were equal and reactive to light and fundoscopy examination was entirely normal. Cranial nerve examination appeared normal. Examining the peripheral nervous system revealed pyramidal weakness and increased tone with brisk reflexes of the right arm and leg. Right plantar reflex was extensor. There was also some reduction in sensation to all modalities affecting the right leg and arm, although this was difficult to test.

Blood tests showed:

Haemoglobin	124 g/L	(115-165)
White cell count	5.6 $\times 10^9$/L	(4-11)
Platelets	276 $\times 10^9$/L	(150-400)
Antinuclear antibodies	1:80	-
Double-stranded DNA antibodies	Negative	-
Erythrocyte sedimentation rate	10 mm/1st hour	(0-30)
C-reactive protein	<5 mg/L	-

Lumbar puncture showed:

Opening pressure	110 mm H_2O	(50-180)
CSF protein	0.28 g/L	(0.15-0.45)
CSF white cell count	14 per ml	(>90% Lymphocytes) ($\leq$ 5)
CSF red cell count	3 per ml	-
CSF glucose	3.3 mmol/L per ml	(3.3-4.4)
CSF oligoclonal bands	Present	-
CSF culture	Negative	-
Serum oligoclonal bands	Absent	-

MR brain scan showed multiple hyperintense lesions affecting paraventricular deep white matter within both cerebral hemispheres. Lesions enhance with gadolinium contrast.

Given the clinical history and investigations so far, what is the likely diagnosis in this patient?

(Please select 1 option)

○ CNS vasculitis

○ Systemic lupus erythematosus (SLE)

○ Herpes simplex encephalitis (HSE)

○ Multiple sclerosis

○ Acute disseminated encephalomyelitis (ADEM)

Submit Answer

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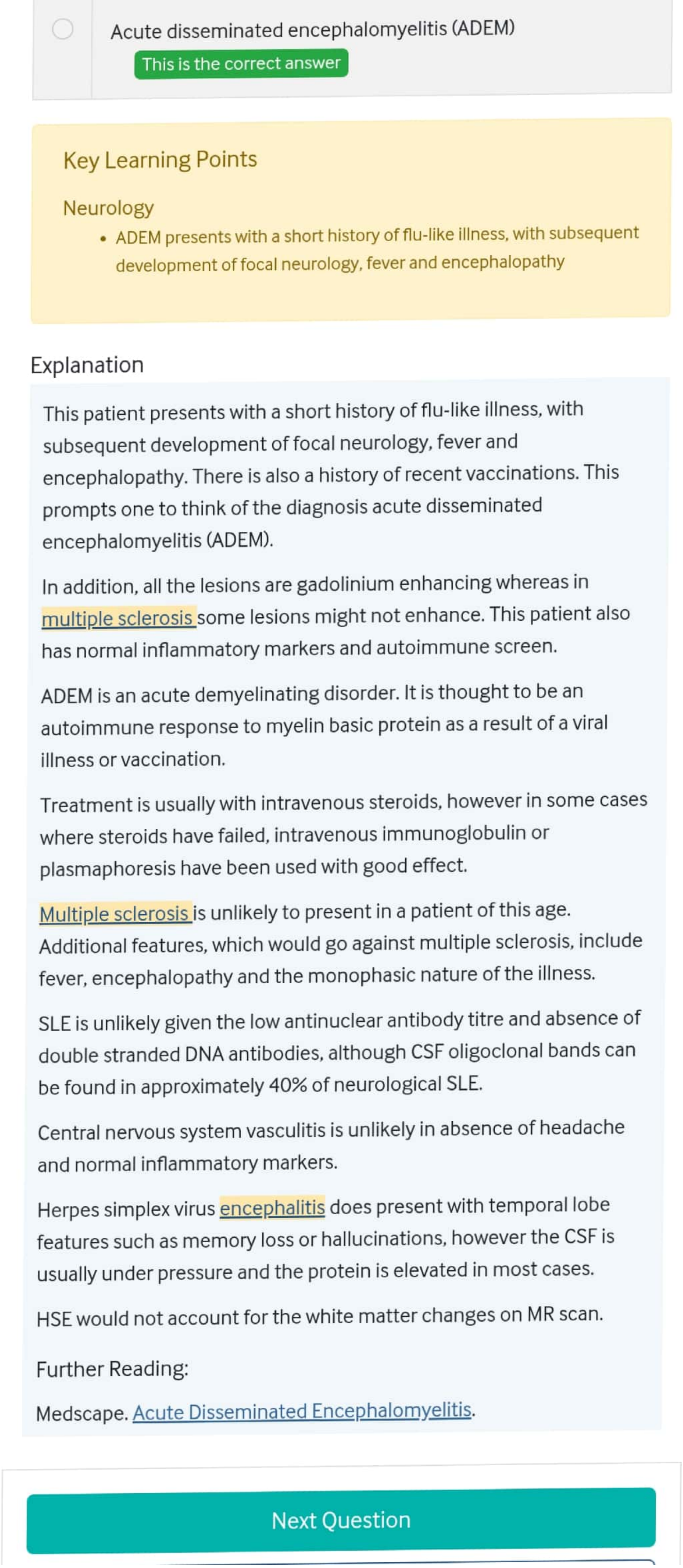


○ Acute disseminated encephalomyelitis (ADEM)

This is the correct answer

Key Learning Points

Neurology

- ADEM presents with a short history of flu-like illness, with subsequent development of focal neurology, fever and encephalopathy

Explanation

This patient presents with a short history of flu-like illness, with subsequent development of focal neurology, fever and encephalopathy. There is also a history of recent vaccinations. This prompts one to think of the diagnosis acute disseminated encephalomyelitis (ADEM).

In addition, all the lesions are gadolinium enhancing whereas in multiple sclerosis some lesions might not enhance. This patient also has normal inflammatory markers and autoimmune screen.

ADEM is an acute demyelinating disorder. It is thought to be an autoimmune response to myelin basic protein as a result of a viral illness or vaccination.

Treatment is usually with intravenous steroids, however in some cases where steroids have failed, intravenous immunoglobulin or plasmaphoresis have been used with good effect.

Multiple sclerosis is unlikely to present in a patient of this age. Additional features, which would go against multiple sclerosis, include fever, encephalopathy and the monophasic nature of the illness.

SLE is unlikely given the low antinuclear antibody titre and absence of double stranded DNA antibodies, although CSF oligoclonal bands can be found in approximately 40% of neurological SLE.

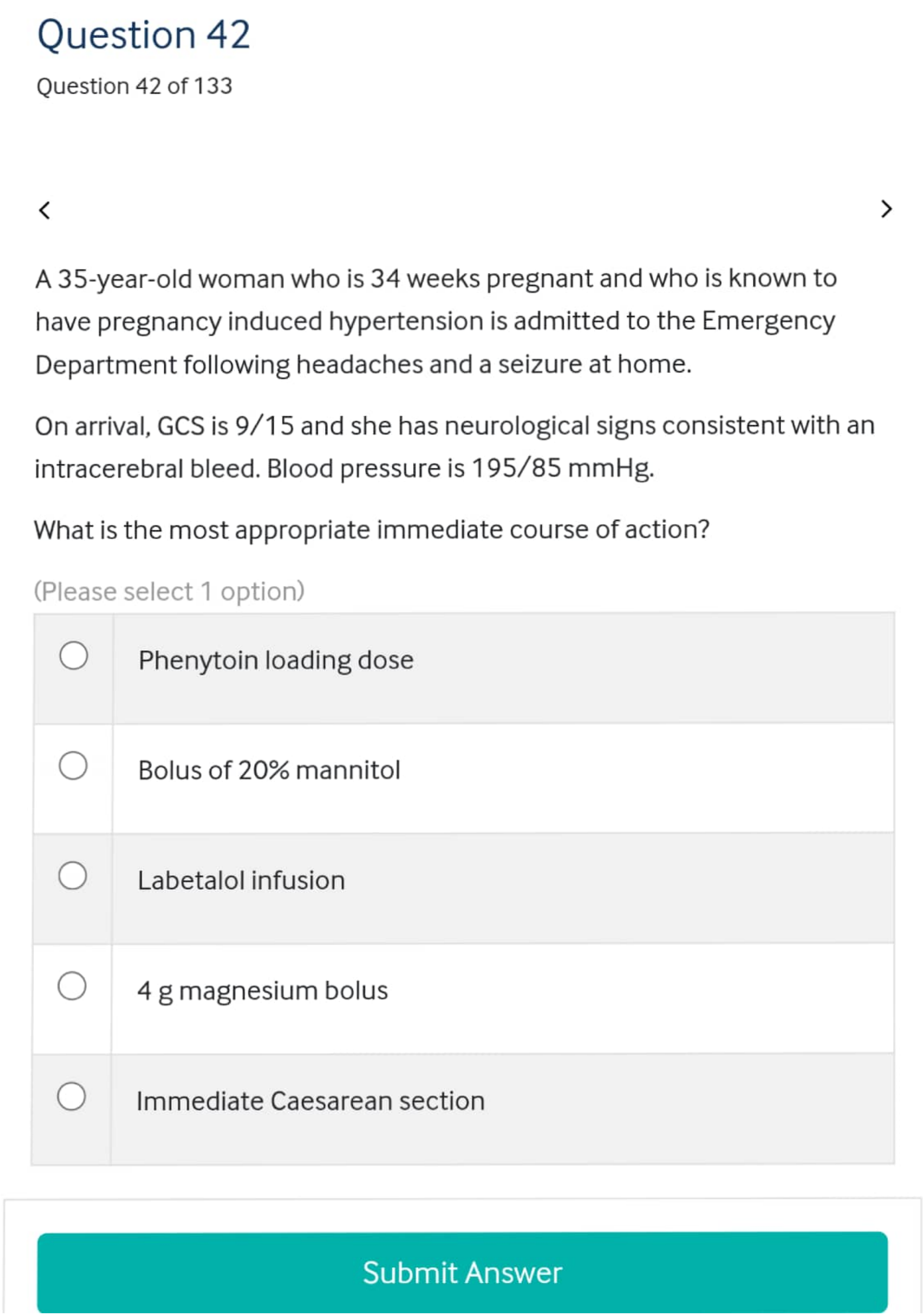
Central nervous system vasculitis is unlikely in absence of headache and normal inflammatory markers.

Herpes simplex virus encephalitis does present with temporal lobe features such as memory loss or hallucinations, however the CSF is usually under pressure and the protein is elevated in most cases.

HSE would not account for the white matter changes on MR scan.

Further Reading:

Medscape. Acute Disseminated Encephalomyelitis.



Question 42

Question 42 of 133

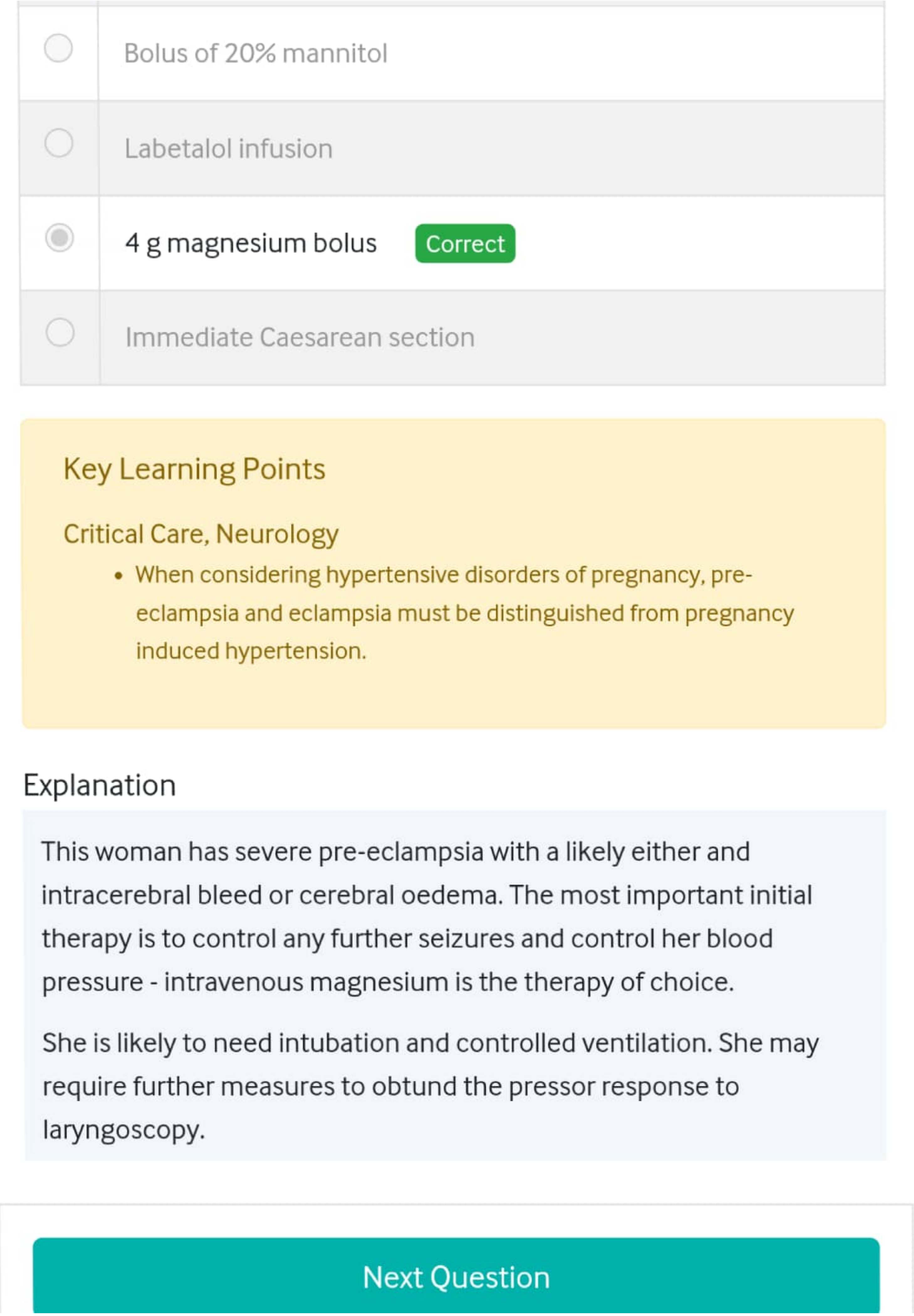
A 35-year-old woman who is 34 weeks pregnant and who is known to have pregnancy induced hypertension is admitted to the Emergency Department following headaches and a seizure at home.

On arrival, GCS is 9/15 and she has neurological signs consistent with an intracerebral bleed. Blood pressure is 195/85 mmHg.

What is the most appropriate immediate course of action?

(Please select 1 option)

- ○ Phenytoin loading dose
- ○ Bolus of 20% mannitol
- ○ Labetalol infusion
- ○ 4 g magnesium bolus
- ○ Immediate Caesarean section



○ Bolus of 20% mannitol

○ Labetalol infusion

● 4 g magnesium bolus **Correct**

○ Immediate Caesarean section

Key Learning Points

Critical Care, Neurology

- When considering hypertensive disorders of pregnancy, pre-eclampsia and eclampsia must be distinguished from pregnancy induced hypertension.

Explanation

This woman has severe pre-eclampsia with a likely either and intracerebral bleed or cerebral oedema. The most important initial therapy is to control any further seizures and control her blood pressure - intravenous magnesium is the therapy of choice.

She is likely to need intubation and controlled ventilation. She may require further measures to obtund the pressor response to laryngoscopy.

A 16-year-old girl is referred to the acute emergency admissions unit by her general practitioner. She attended the GP practice with her mother earlier that day with a three week history of problems with her co-ordinations. Her mother had noticed that she is repeatedly dropping things and more clumsy than usual, for example, walking into furniture, difficulty in using cutlery.

She was previously fit and well with no past medical history of note. She is currently studying for her GCSEs and is hoping to continue on to sixth form to study A-levels. She is physically very active and is a member of the school hockey and netball team. The only family history of note is her father has a history of epilepsy but is well controlled on phenytoin and sodium valproate.

On examination, she looked well nourished and hydrated. A member of nursing staff had recorded a single episode of vomiting since arrival on the admissions unit, which was bile stained. Her pulse rate was 80 beats per min, regular; blood pressure was 110 systolic and 60 diastolic. Cardiovascular, respiratory and abdominal examinations were normal.

On neurological examination she had an ataxic gait, mild tremor of both hands, mild dysarthria, bilateral rapid nystagmus on lateral gaze, bilateral dysdiadochokinesia and exaggerated reflexes throughout but plantars were flexor. Fundoscopy was normal.

Investigations revealed:

Serum ALT	102 U/L	(5-35)
Serum ALP	115 U/L	(45-105)
Serum GGT	163 U/L	(4-35)
Serum total bilirubin	21 μmol/L	(1-22)
Serum calcium	2.57 mmol/L	(2.2-2.6)
Serum albumin	45 g/L	(37-49)
Random glucose	8.1 mmol/L	(<11.1)
Serum ferritin	376 μg/L	(15-200)
Serum caeruloplasmin	0.45 g/L	(0.2-0.6)

A magnetic resonance imaging (MRI) scan was performed and reported as normal.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Sodium valproate toxicity
- ○ Phenytoin toxicity
- ○ Wilson disease
- ○ Herpes simplex encephalitis
- ○ Haemochromatosis

Submit Answer

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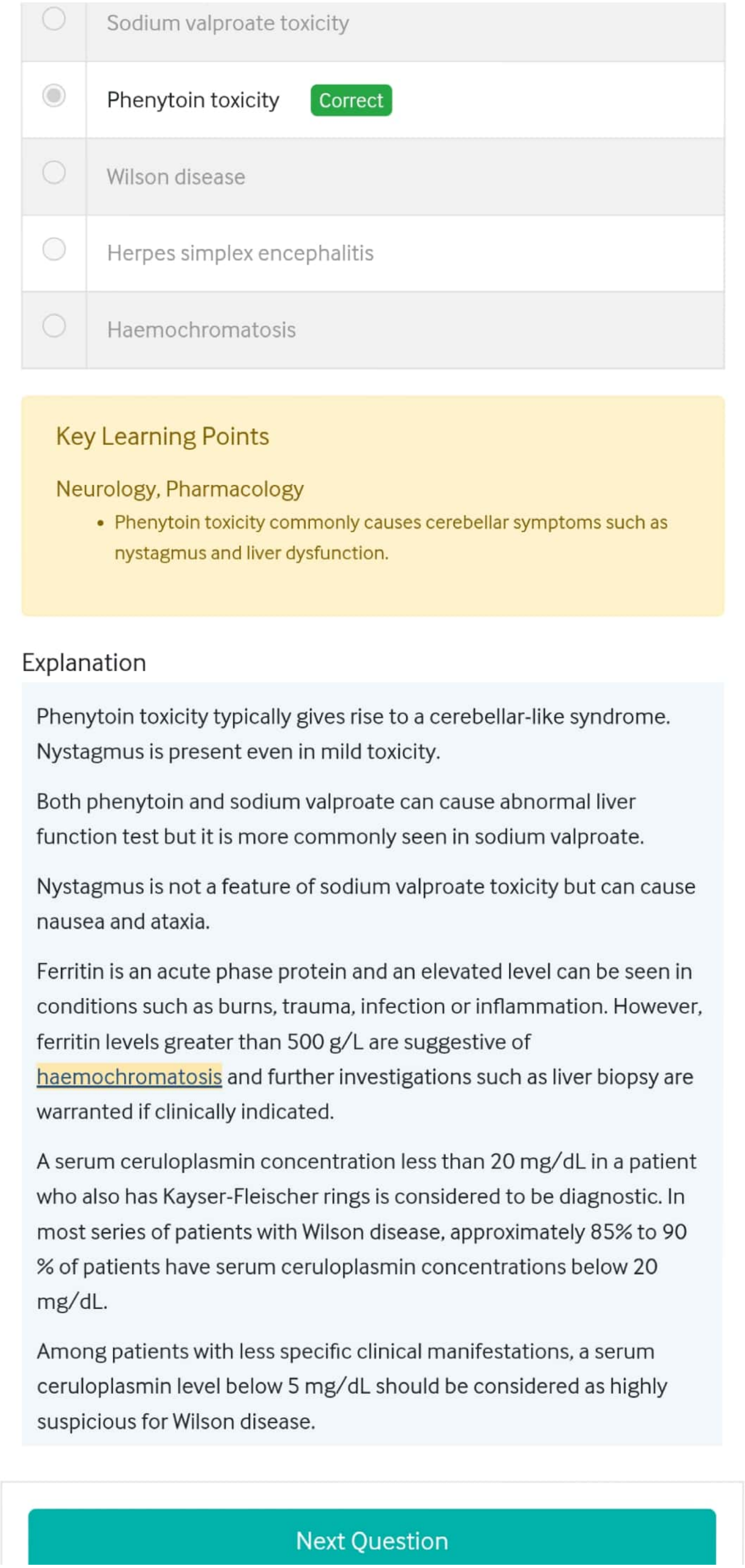


○ Sodium valproate toxicity

● Phenytoin toxicity **Correct**

○ Wilson disease

○ Herpes simplex encephalitis

○ Haemochromatosis

Key Learning Points

Neurology, Pharmacology

- Phenytoin toxicity commonly causes cerebellar symptoms such as nystagmus and liver dysfunction.

Explanation

Phenytoin toxicity typically gives rise to a cerebellar-like syndrome. Nystagmus is present even in mild toxicity.

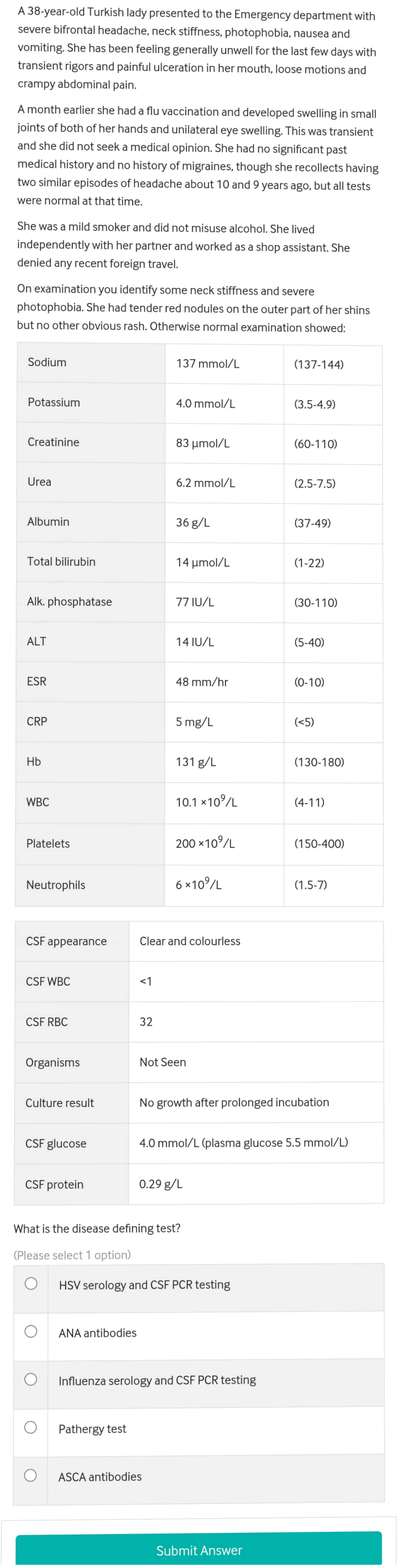
Both phenytoin and sodium valproate can cause abnormal liver function test but it is more commonly seen in sodium valproate.

Nystagmus is not a feature of sodium valproate toxicity but can cause nausea and ataxia.

Ferritin is an acute phase protein and an elevated level can be seen in conditions such as burns, trauma, infection or inflammation. However, ferritin levels greater than 500 g/L are suggestive of haemochromatosis and further investigations such as liver biopsy are warranted if clinically indicated.

A serum ceruloplasmin concentration less than 20 mg/dL in a patient who also has Kayser-Fleischer rings is considered to be diagnostic. In most series of patients with Wilson disease, approximately 85% to 90 % of patients have serum ceruloplasmin concentrations below 20 mg/dL.

Among patients with less specific clinical manifestations, a serum ceruloplasmin level below 5 mg/dL should be considered as highly suspicious for Wilson disease.



A 38-year-old Turkish lady presented to the Emergency department with severe bifrontal headache, neck stiffness, photophobia, nausea and vomiting. She has been feeling generally unwell for the last few days with transient rigors and painful ulceration in her mouth, loose motions and crampy abdominal pain.

A month earlier she had a flu vaccination and developed swelling in small joints of both of her hands and unilateral eye swelling. This was transient and she did not seek a medical opinion. She had no significant past medical history and no history of migraines, though she recollects having two similar episodes of headache about 10 and 9 years ago, but all tests were normal at that time.

She was a mild smoker and did not misuse alcohol. She lived independently with her partner and worked as a shop assistant. She denied any recent foreign travel.

On examination you identify some neck stiffness and severe photophobia. She had tender red nodules on the outer part of her shins but no other obvious rash. Otherwise normal examination showed:

Sodium	137 mmol/L	(137-144)
Potassium	4.0 mmol/L	(3.5-4.9)
Creatinine	83 µmol/L	(60-110)
Urea	6.2 mmol/L	(2.5-7.5)
Albumin	36 g/L	(37-49)
Total bilirubin	14 µmol/L	(1-22)
Alk. phosphatase	77 IU/L	(30-110)
ALT	14 IU/L	(5-40)
ESR	48 mm/hr	(0-10)
CRP	5 mg/L	(<5)
Hb	131 g/L	(130-180)
WBC	10.1 ×10^9/L	(4-11)
Platelets	200 ×10^9/L	(150-400)
Neutrophils	6 ×10^9/L	(1.5-7)

CSF appearance	Clear and colourless
CSF WBC	<1
CSF RBC	32
Organisms	Not Seen
Culture result	No growth after prolonged incubation
CSF glucose	4.0 mmol/L (plasma glucose 5.5 mmol/L)
CSF protein	0.29 g/L

What is the disease defining test?

(Please select 1 option)

○ HSV serology and CSF PCR testing

○ ANA antibodies

○ Influenza serology and CSF PCR testing

○ Pathergy test

○ ASCA antibodies

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● Pathergy test **Correct**

○ ASCA antibodies

Key Learning Points

Neurology

- Bechet's disease will produce a positive pethergy test.

Explanation

Pathergy reaction elicited by performing a skin prick using a sterile needle and waiting 48 hours to see whether a papule or pustule forms is one of the diagnostic criteria for Behçet's syndrome which may cause aseptic meningitis.

Behçet's syndrome is a systemic vasculitis that leads to mucocutaneous, ophthalmological, vascular, gastrointestinal, and central nervous system (CNS) manifestations. It is most commonly seen in young people aged 20 to 30 years from the Middle East, the Mediterranean region, and the Far East.

It is more active and severe in males. Clinical presentation is typical and is the base for the diagnosis. Recurrent mucocutanoeus ulcerations are typical. Other manifestations are:

- Eye - uveitis, hypopyon
- Skin - acneiform lesions in arms and legs, erythema nodosum, superficial thrombophlebitis
- Gastroinstestinal - diarrhoea, cramping abdominal pain, and mucosal ulcerations
- Major vessel - thrombophlebitis, DVT, dural sinus thrombosis, pulmonary aneurysm
- CNS - aseptic meningitis, memory impairment. Impaired speech, balance, and movement are possible.

MRI findings in the brain can be similar to those in multiple sclerosis in some cases or there may be distinct white matter changes commonly involving the midbrain. Azathioprine and other immunosuppressive therapies are used when severe manifestations such as eye, vascular, and CNS involvement are present.

Some of the other causes for aseptic meningitis are:

- viruses - commonly enteroviruses, e.g. polio, coxsackievirus, echovirus
- partially treated bacterial meningitis
- fungal meningitis
- parasites
- drugs, e.g. intravenous immunoglobulin, and some intrathecal medications, and
- systemic diseases.

The clinical presentation is typical enough to make a reasonable hypothesis for the diagnosis. Further, cerebrospinal fluid (CSF) examination showing normal microscopy and biochemistry essentially rules out the possibility of viral or bacterial origin in this scenario.

In viral meningitis, CSF WBC count may be normal but typically is above 5 cells/mm^3. A CSF WBC count above 5 cells/mm^3 has a sensitivity of 90% for the diagnosis of viral meningitis.

Polymerase chain reaction (PCR) for herpes viruses has sensitivity and specificity of greater than 95% for all herpes CNS infections. This is not specific for meningitis. In this scenario, given the initial CSF screen is negative, this would not be done unless there was strong clinical suspicion.

Case reports describe various post-flu vaccination neurological complications, ranging from mild myalgia to severe encephalomyelitis or transverse myelitis with subsequent seizure or paraplegia presentation.

The history of flu vaccination is misleading here. Although it can also cause aseptic meningitis, the Behçet's syndrome is much more likely given the clinical characteristics.

Rheumatoid factor, anti-nuclear antibodies, and anti-neutrophil cytoplasmic antibodies are usually negative in Behçet's syndrome.

Anti-saccharomyces cerevisiae antibodies may be elevated but further studies are needed.

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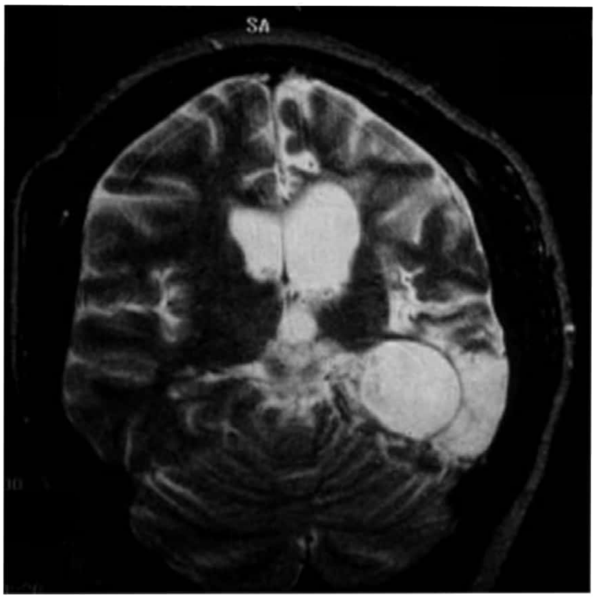
A 35-year-old woman is brought to the Emergency Department by her husband.

She is drowsy and confused. There is a three day history of malaise and low-grade fever, but she has become increasingly confused over the past eight hours and has apparently been drifting in and out of conciousness.

On examination she is drowsy but rousable. GCS 8/15. There are no focal neurological signs. While in the emergency unit she has a generalised tonic-clonic seizure.

An MRI scan of her brain is shown.



A lumbar puncture was performed and analysis of the cerebrospinal fluid revealed:

Appearance	Clear, colourless	
Cells	500/mm^3 (>90% lymphocytes)	(60-70)
Protein	0.7 g/L	(0.15-0.45)
Glucose	4.5 mmol/L	(3.3-4.4)

Plasma glucose was measured as 5.3 mmol/L.

Which of the following investigations is most likely to reveal the correct diagnosis?

(Please select 1 option)

○ CSF for *Cryptococcus neoformans*

○ Blood cultures

○ Magnetic resonance angiogram

○ CSF for HSV PCR

○ Serum for viral titres

Submit Answer

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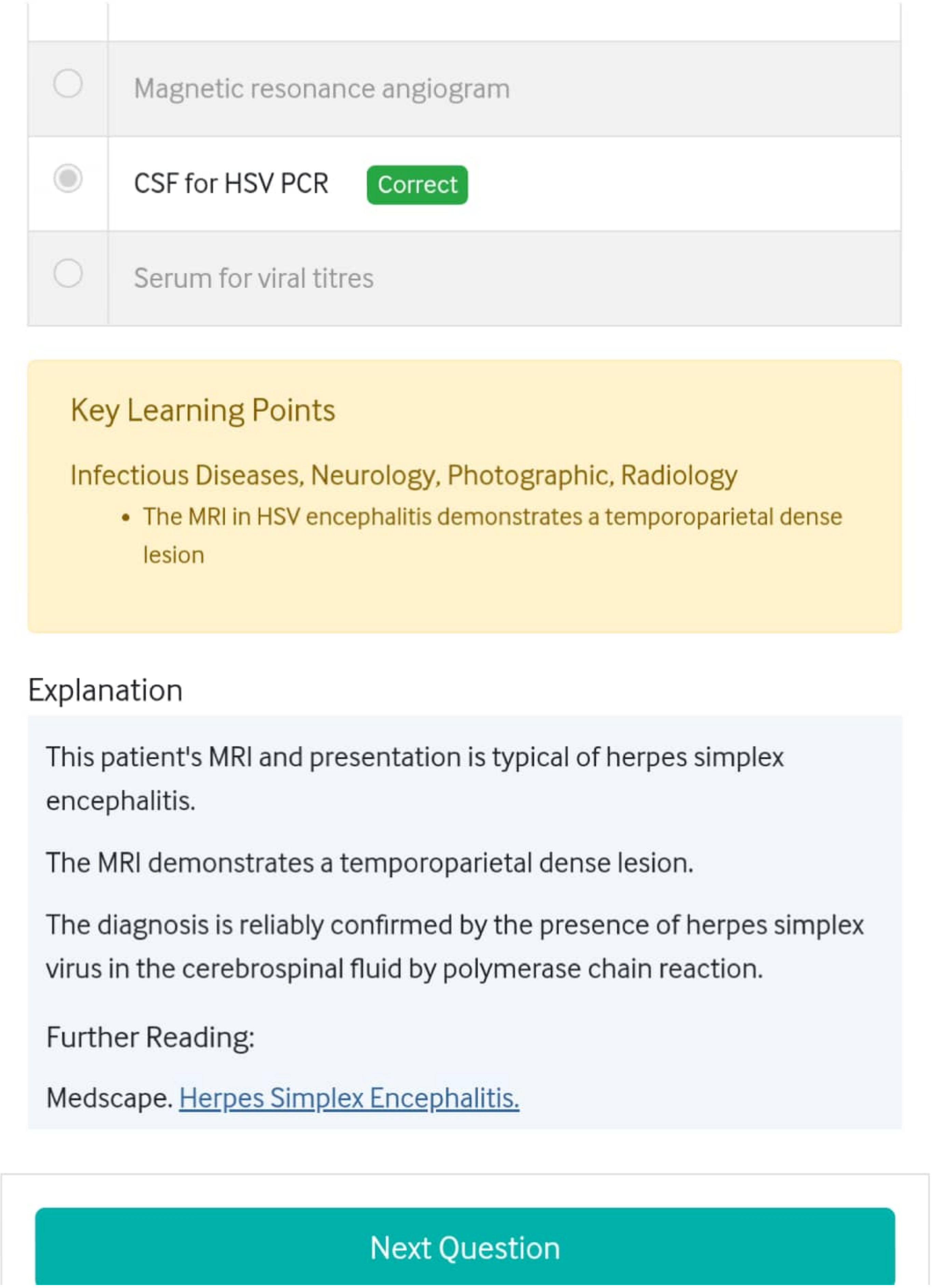


○ Magnetic resonance angiogram

● CSF for HSV PCR **Correct**

○ Serum for viral titres

Key Learning Points

Infectious Diseases, Neurology, Photographic, Radiology

- The MRI in HSV encephalitis demonstrates a temporoparietal dense lesion

Explanation

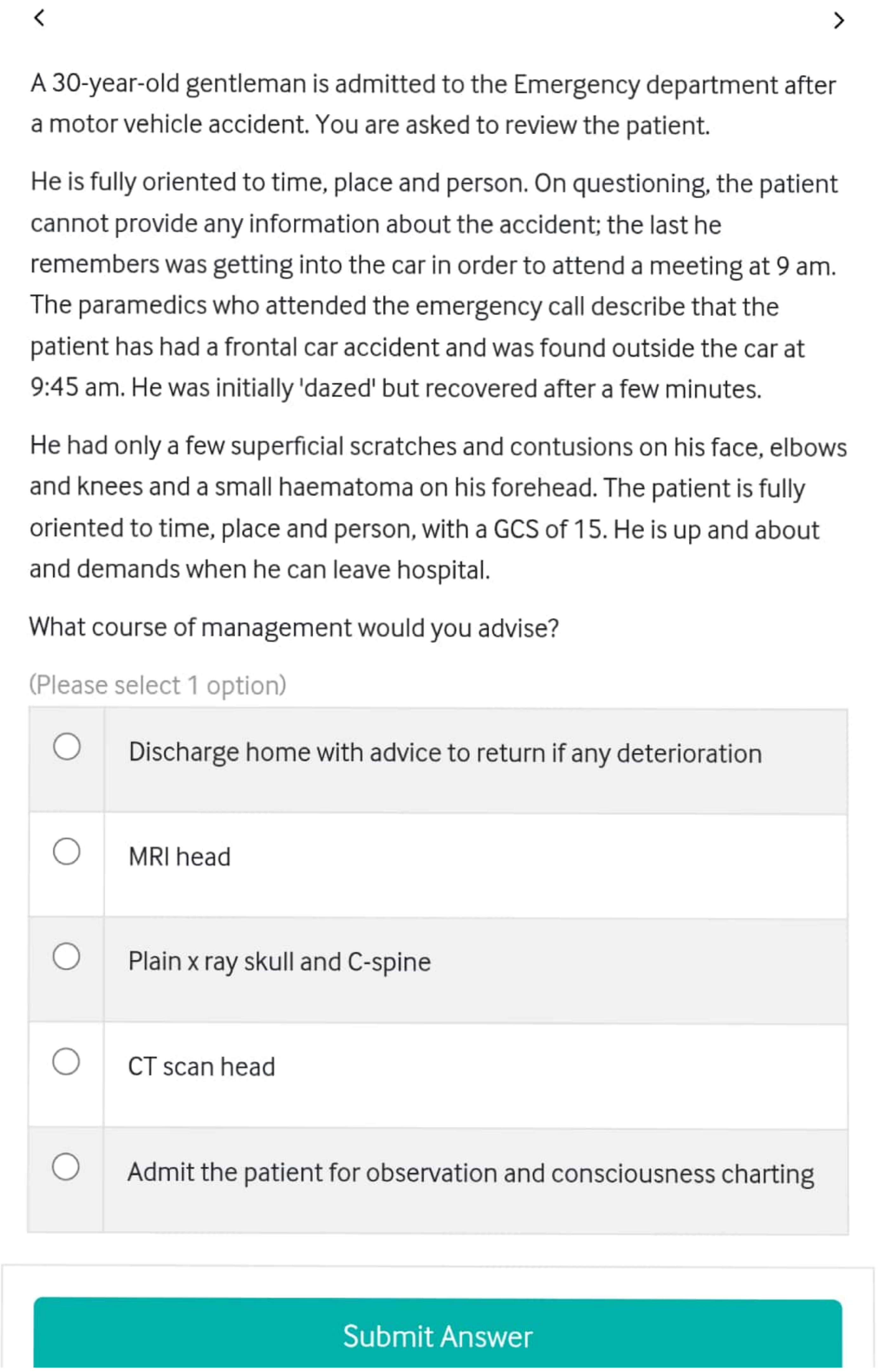
This patient's MRI and presentation is typical of herpes simplex encephalitis.

The MRI demonstrates a temporoparietal dense lesion.

The diagnosis is reliably confirmed by the presence of herpes simplex virus in the cerebrospinal fluid by polymerase chain reaction.

Further Reading:

Medscape. Herpes Simplex Encephalitis.



A 30-year-old gentleman is admitted to the Emergency department after a motor vehicle accident. You are asked to review the patient.

He is fully oriented to time, place and person. On questioning, the patient cannot provide any information about the accident; the last he remembers was getting into the car in order to attend a meeting at 9 am. The paramedics who attended the emergency call describe that the patient has had a frontal car accident and was found outside the car at 9:45 am. He was initially 'dazed' but recovered after a few minutes.

He had only a few superficial scratches and contusions on his face, elbows and knees and a small haematoma on his forehead. The patient is fully oriented to time, place and person, with a GCS of 15. He is up and about and demands when he can leave hospital.

What course of management would you advise?

(Please select 1 option)

- ○ Discharge home with advice to return if any deterioration
- ○ MRI head
- ○ Plain x ray skull and C-spine
- ○ CT scan head
- ○ Admit the patient for observation and consciousness charting

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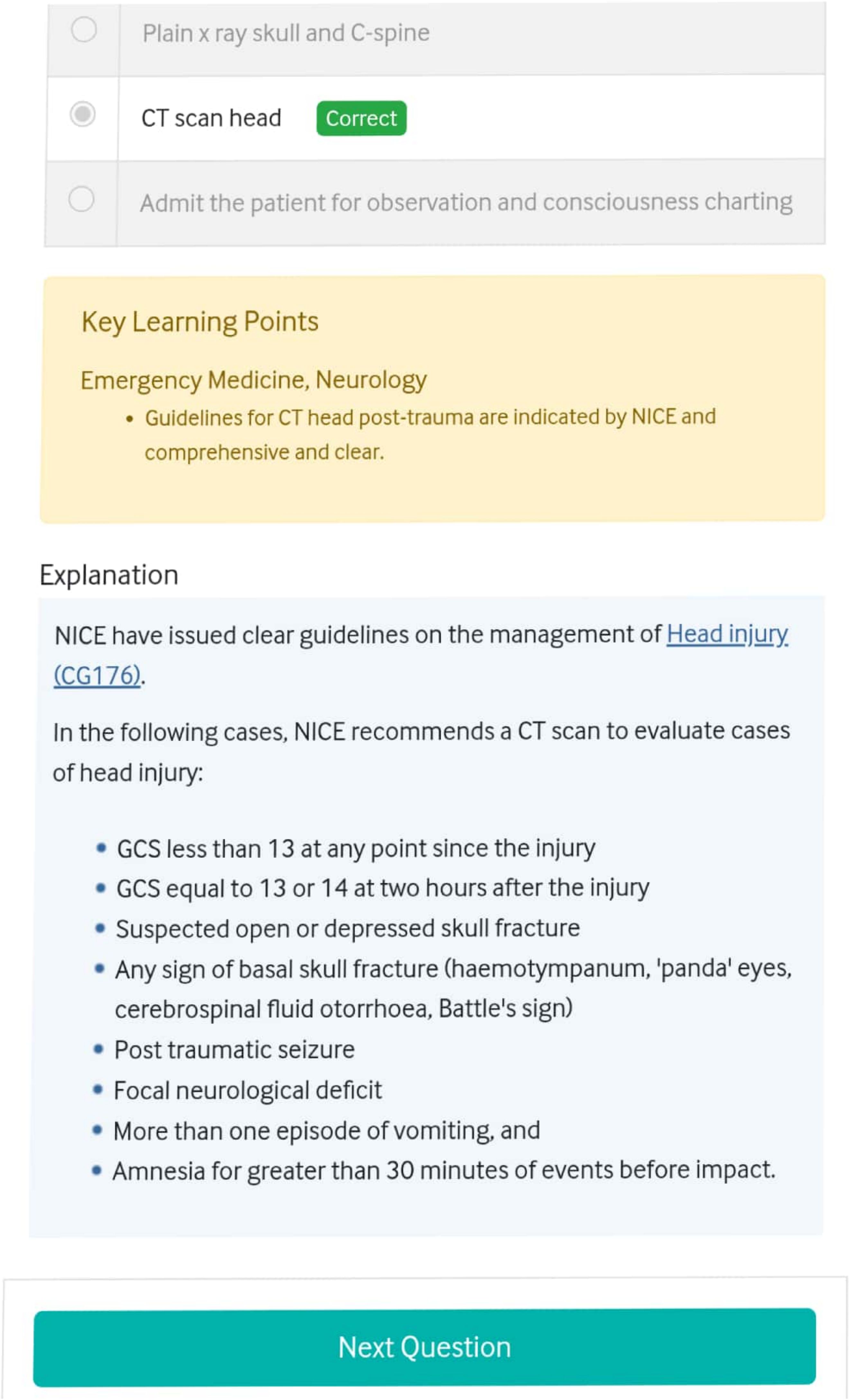


○ Plain x ray skull and C-spine

● CT scan head **Correct**

○ Admit the patient for observation and consciousness charting

Key Learning Points

Emergency Medicine, Neurology

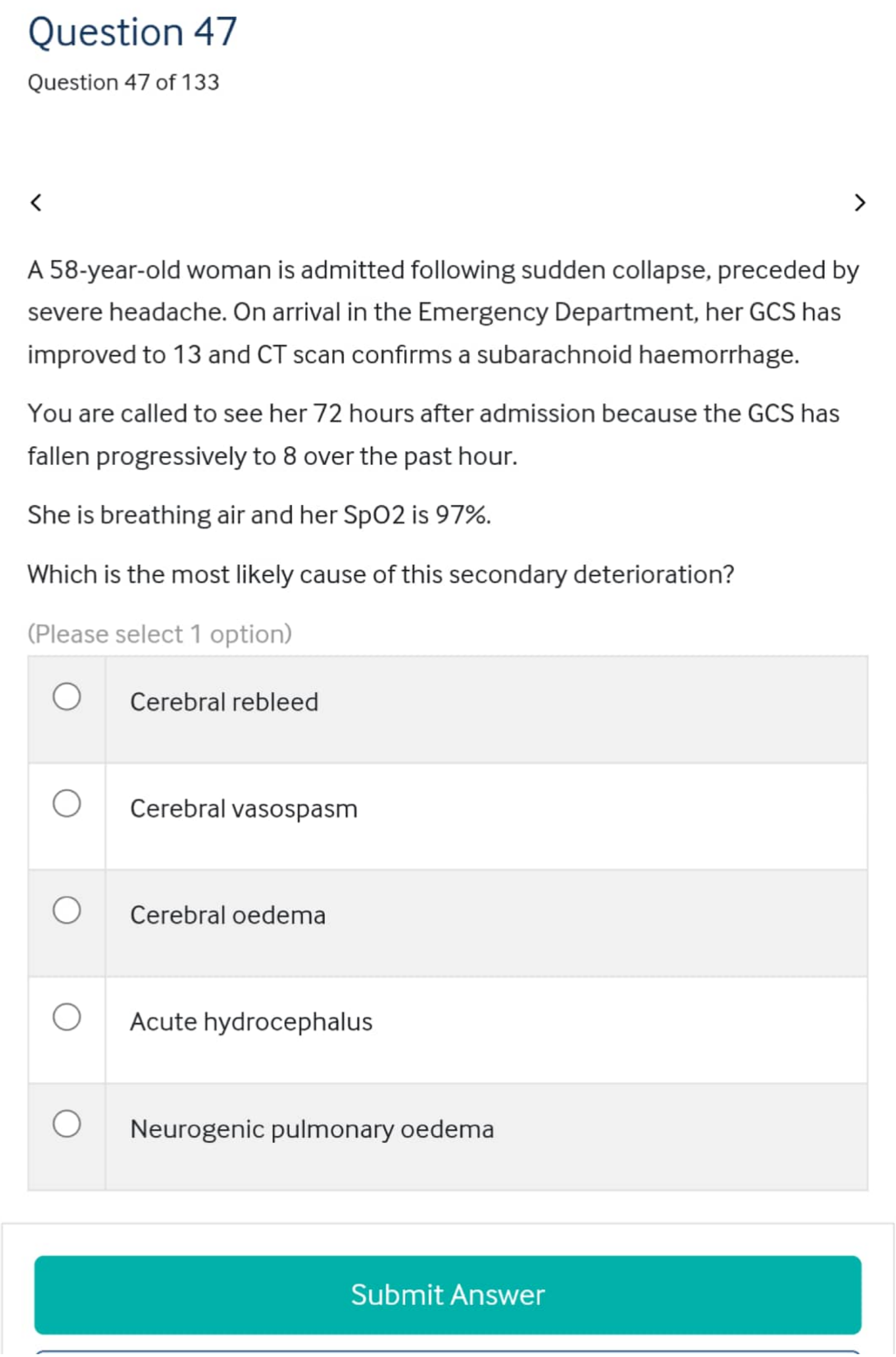
- Guidelines for CT head post-trauma are indicated by NICE and comprehensive and clear.

Explanation

NICE have issued clear guidelines on the management of Head injury (CG176).

In the following cases, NICE recommends a CT scan to evaluate cases of head injury:

- GCS less than 13 at any point since the injury
- GCS equal to 13 or 14 at two hours after the injury
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid otorrhoea, Battle's sign)
- Post traumatic seizure
- Focal neurological deficit
- More than one episode of vomiting, and
- Amnesia for greater than 30 minutes of events before impact.



Question 47

Question 47 of 133

A 58-year-old woman is admitted following sudden collapse, preceded by severe headache. On arrival in the Emergency Department, her GCS has improved to 13 and CT scan confirms a subarachnoid haemorrhage.

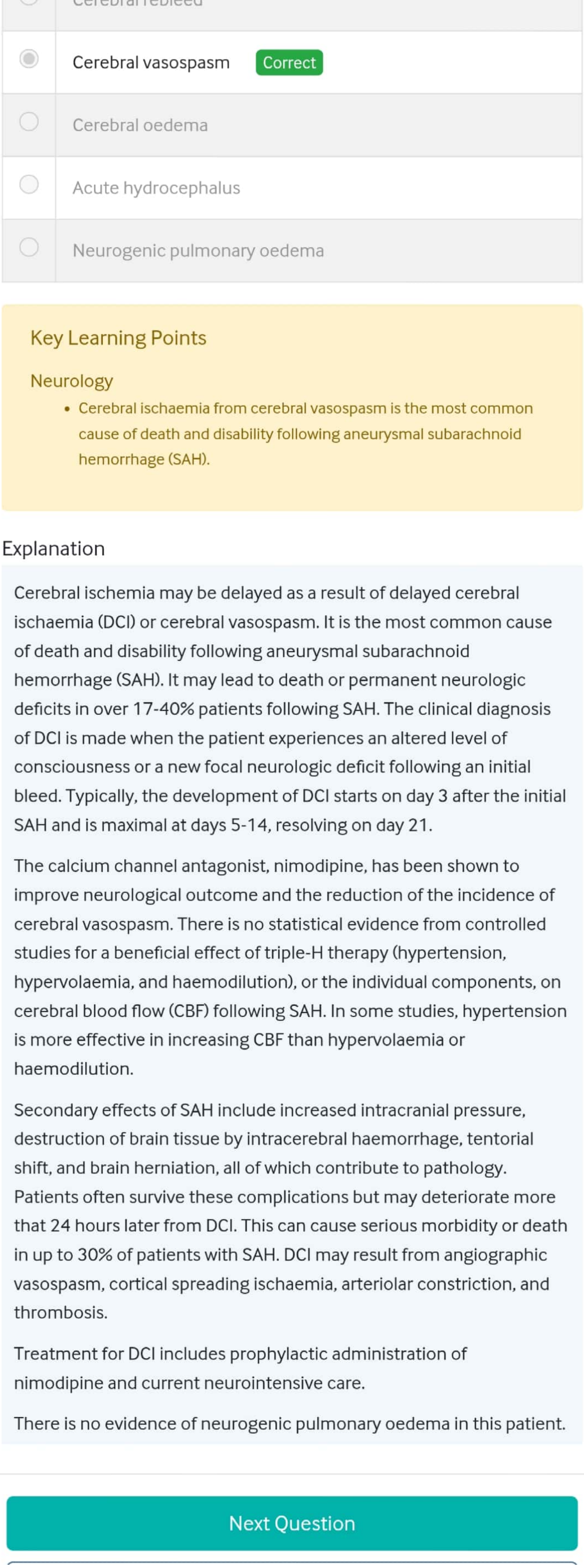
You are called to see her 72 hours after admission because the GCS has fallen progressively to 8 over the past hour.

She is breathing air and her SpO2 is 97%.

Which is the most likely cause of this secondary deterioration?

(Please select 1 option)

- ○ Cerebral rebleed
- ○ Cerebral vasospasm
- ○ Cerebral oedema
- ○ Acute hydrocephalus
- ○ Neurogenic pulmonary oedema



○ Cerebral rebleed

● Cerebral vasospasm Correct

○ Cerebral oedema

○ Acute hydrocephalus

○ Neurogenic pulmonary oedema

Key Learning Points

Neurology

- Cerebral ischaemia from cerebral vasospasm is the most common cause of death and disability following aneurysmal subarachnoid hemorrhage (SAH).

Explanation

Cerebral ischemia may be delayed as a result of delayed cerebral ischaemia (DCI) or cerebral vasospasm. It is the most common cause of death and disability following aneurysmal subarachnoid hemorrhage (SAH). It may lead to death or permanent neurologic deficits in over 17-40% patients following SAH. The clinical diagnosis of DCI is made when the patient experiences an altered level of consciousness or a new focal neurologic deficit following an initial bleed. Typically, the development of DCI starts on day 3 after the initial SAH and is maximal at days 5-14, resolving on day 21.

The calcium channel antagonist, nimodipine, has been shown to improve neurological outcome and the reduction of the incidence of cerebral vasospasm. There is no statistical evidence from controlled studies for a beneficial effect of triple-H therapy (hypertension, hypervolaemia, and haemodilution), or the individual components, on cerebral blood flow (CBF) following SAH. In some studies, hypertension is more effective in increasing CBF than hypervolaemia or haemodilution.

Secondary effects of SAH include increased intracranial pressure, destruction of brain tissue by intracerebral haemorrhage, tentorial shift, and brain herniation, all of which contribute to pathology. Patients often survive these complications but may deteriorate more that 24 hours later from DCI. This can cause serious morbidity or death in up to 30% of patients with SAH. DCI may result from angiographic vasospasm, cortical spreading ischaemia, arteriolar constriction, and thrombosis.

Treatment for DCI includes prophylactic administration of nimodipine and current neurointensive care.

There is no evidence of neurogenic pulmonary oedema in this patient.

CamScanner المسوحة ضوئيا بـ

A 22-year-old epileptic woman was admitted to the Emergency department with a three day history of worsening generalised headache, associated with vomiting, unsteadiness and blurred vision. The unsteadiness had progressively worsened over six weeks, and in the last two weeks her speech had become slurred. She had lost one stone in weight within the last month, although attributed this to pressure at work, as she had felt increasingly nauseated and tired.

She had epilepsy, which was diagnosed at the age of 6 and had been under the ophthalmologists in the past for multiple angiomas of the left eye. Her current medication included phenytoin, carbamazepine and the oral contraceptive pill.

On examination she appeared unwell and had marked dysarthria. Her blood pressure was 150/89 mmHg and pulse 69/minute. Fundoscopy examination revealed bilateral papilloedema and evidence of previous photocoagulation. She had reduced visual acuity of 6/18 in both eyes, however pupillary responses were normal. The rest of the cranial nerves were normal.

On examination of the upper and lower limb, tone, power and reflexes all appeared normal, however there was a marked bilateral intention tremor and past pointing. She had a broad based and ataxic gait. Sensory examination appeared normal. Chest examination appeared normal, however on abdominal examination there was a ballotable right kidney.

Investigations revealed:

Haemoglobin	185 g/L	(115-165)
Packed cell volume	0.55	(0.37-0.47)
Red cell count	7.0×10^{12}/L	(3.9-5.6)
Mean cell volume	88 fL	(80-96)
White cell count	9.8×10^{9}/L	(4-11)
Platelets	399×10^{9}/L	(150-400)
Serum sodium	133 mmol/L	(137-144)
Serum potassium	4.5 mmol/L	(3.5-4.9)
Serum urea	8.0 mmol/L	(2.5-7.5)
Serum creatinine	220 mol/L	(60-110)
Serum calcium	2.7 mmol/L	(2.2-2.6)
Phenytoin level	81 mol/L	(therapeutic range 40-80)

Urinalysis: Blood +++ Protein ++

An MRI scan of the brain was reported as showing bilateral cerebellar haemangiomas.

What is the most likely underlying diagnosis in this patient?

(Please select 1 option)

○ Neurofibromatosis type 2

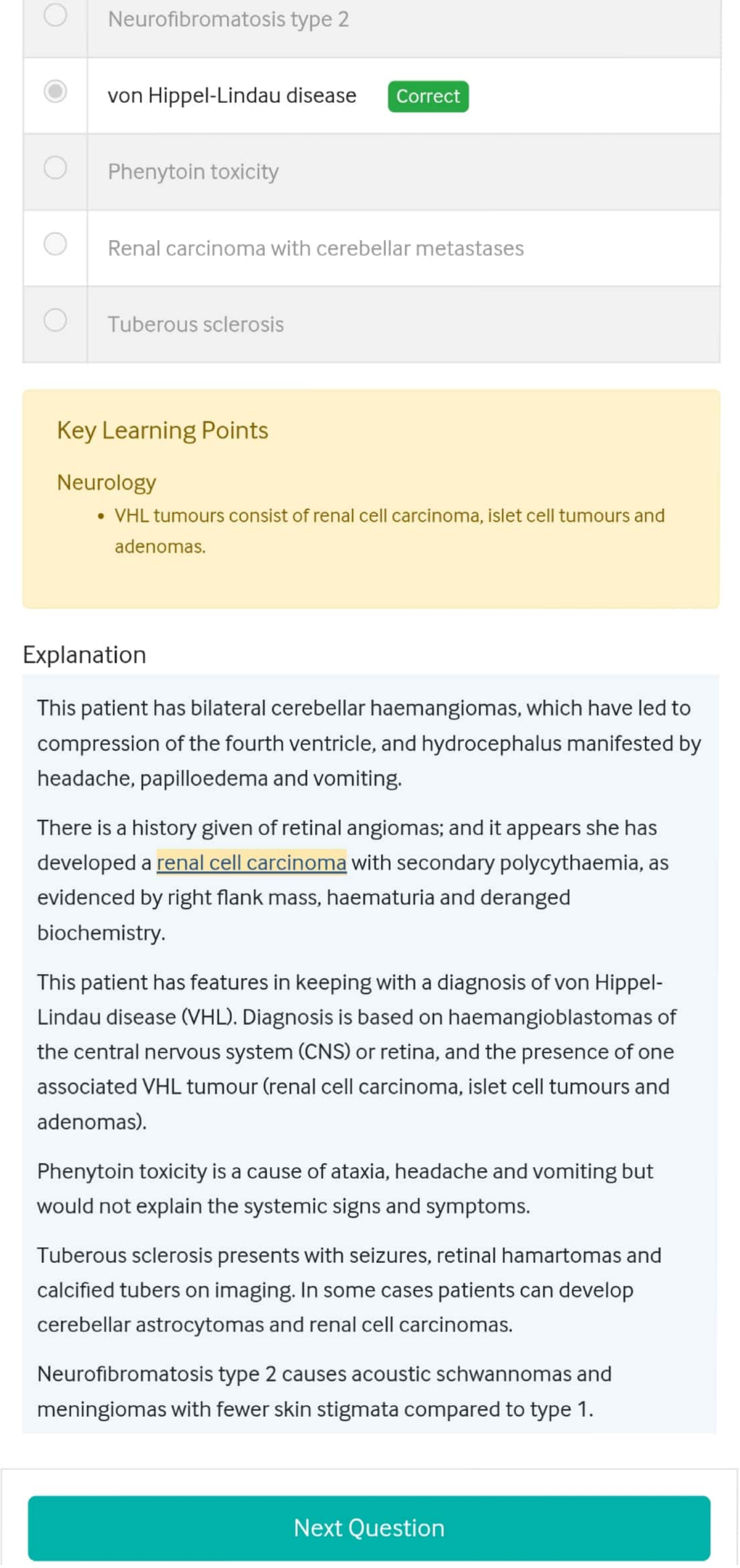
○ von Hippel-Lindau disease

○ Phenytoin toxicity

○ Renal carcinoma with cerebellar metastases

○ Tuberous sclerosis

Submit Answer



○ Neurofibromatosis type 2

● von Hippel-Lindau disease Correct

○ Phenytoin toxicity

○ Renal carcinoma with cerebellar metastases

○ Tuberous sclerosis

Key Learning Points

Neurology

- VHL tumours consist of renal cell carcinoma, islet cell tumours and adenomas.

Explanation

This patient has bilateral cerebellar haemangiomas, which have led to compression of the fourth ventricle, and hydrocephalus manifested by headache, papilloedema and vomiting.

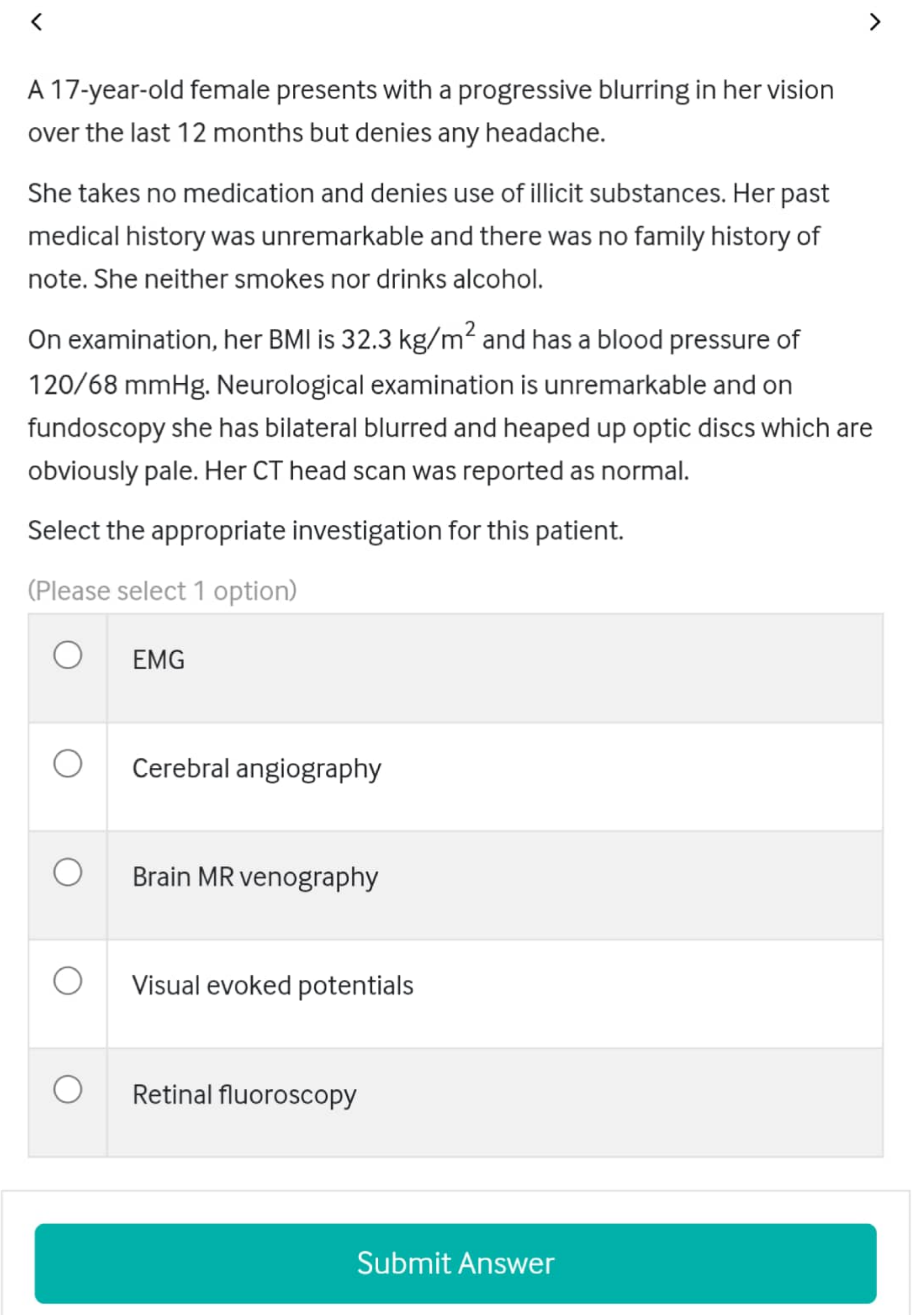
There is a history given of retinal angiomas; and it appears she has developed a renal cell carcinoma with secondary polycythaemia, as evidenced by right flank mass, haematuria and deranged biochemistry.

This patient has features in keeping with a diagnosis of von Hippel-Lindau disease (VHL). Diagnosis is based on haemangioblastomas of the central nervous system (CNS) or retina, and the presence of one associated VHL tumour (renal cell carcinoma, islet cell tumours and adenomas).

Phenytoin toxicity is a cause of ataxia, headache and vomiting but would not explain the systemic signs and symptoms.

Tuberous sclerosis presents with seizures, retinal hamartomas and calcified tubers on imaging. In some cases patients can develop cerebellar astrocytomas and renal cell carcinomas.

Neurofibromatosis type 2 causes acoustic schwannomas and meningiomas with fewer skin stigmata compared to type 1.



A 17-year-old female presents with a progressive blurring in her vision over the last 12 months but denies any headache.

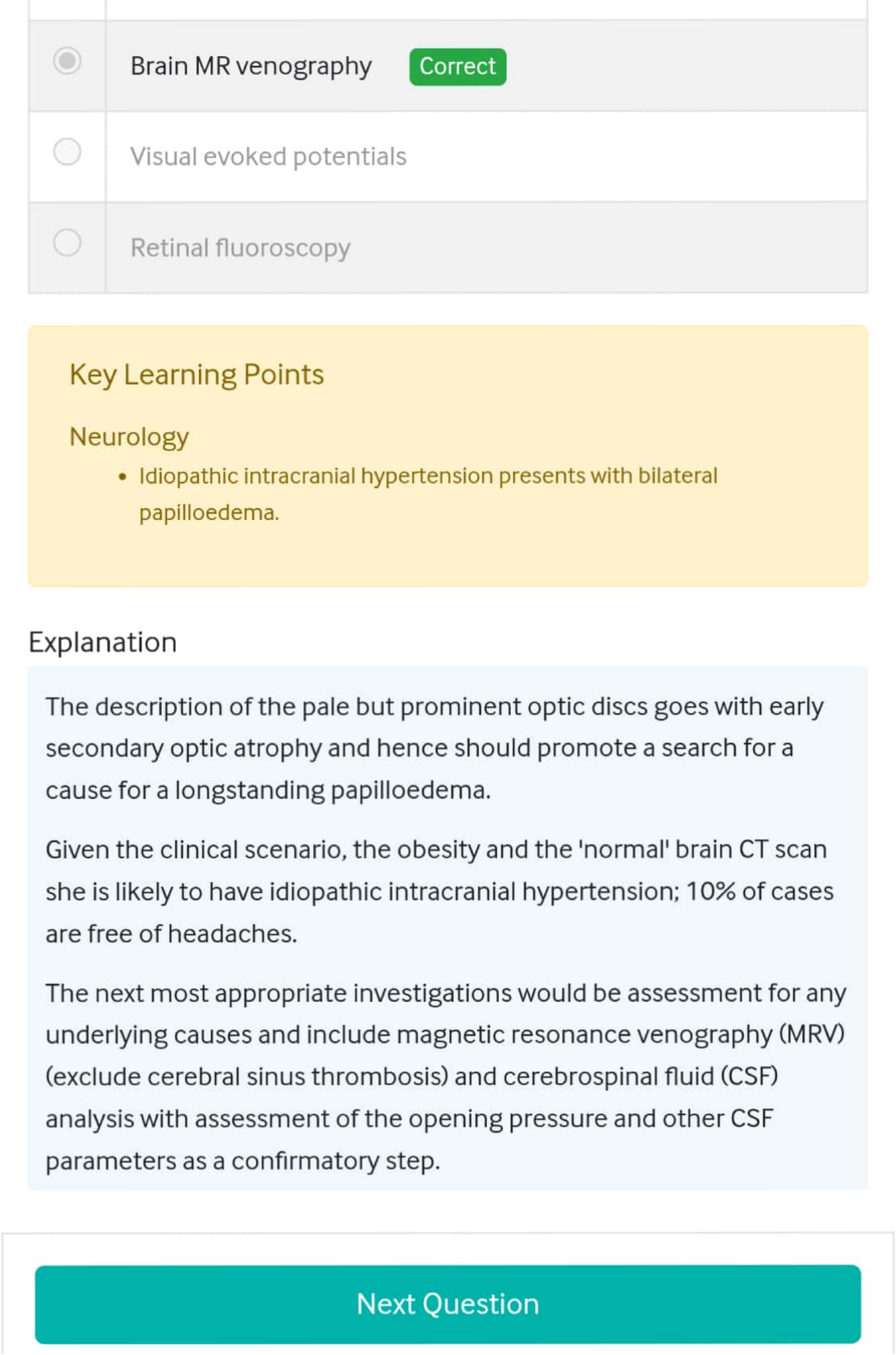
She takes no medication and denies use of illicit substances. Her past medical history was unremarkable and there was no family history of note. She neither smokes nor drinks alcohol.

On examination, her BMI is 32.3 kg/m^2 and has a blood pressure of 120/68 mmHg. Neurological examination is unremarkable and on fundoscopy she has bilateral blurred and heaped up optic discs which are obviously pale. Her CT head scan was reported as normal.

Select the appropriate investigation for this patient.

(Please select 1 option)

- ○ EMG
- ○ Cerebral angiography
- ○ Brain MR venography
- ○ Visual evoked potentials
- ○ Retinal fluoroscopy



● Brain MR venography **Correct**

○ Visual evoked potentials

○ Retinal fluoroscopy

Key Learning Points

Neurology

- Idiopathic intracranial hypertension presents with bilateral papilloedema.

Explanation

The description of the pale but prominent optic discs goes with early secondary optic atrophy and hence should promote a search for a cause for a longstanding papilloedema.

Given the clinical scenario, the obesity and the 'normal' brain CT scan she is likely to have idiopathic intracranial hypertension; 10% of cases are free of headaches.

The next most appropriate investigations would be assessment for any underlying causes and include magnetic resonance venography (MRV) (exclude cerebral sinus thrombosis) and cerebrospinal fluid (CSF) analysis with assessment of the opening pressure and other CSF parameters as a confirmatory step.

‹ ›

A 56-year-old female was seen by her GP complaining of lethargy and generalised muscle weakness. She had noticed marked difficulty in combing her hair and walking down stairs.

There was no associated muscle ache or tenderness and she did not complain of any neck pain. She had a long-standing history of severe rheumatoid arthritis for which she took regular steroids and penicillamine. She had no other medical history, was a smoker of 10 cigarettes per day and did not drink alcohol.

On examination she appeared apathetic and there was evidence of bilateral ptosis. Cranial nerve examination revealed oculoparesis of the right lateral rectus and left medial rectus, with normal pupillary responses. There was some mild facial weakness, but she had normal palatal movements and her speech was normal.

On examination of the upper limb, there was some weakness of neck extension and shoulder abduction and adduction. Distal power was normal as were reflexes, tone and sensation. Lower limb examination revealed peripheral oedema and weakness of hip flexion and extension with preserved distal power, reflexes, tone and sensation. Neck examination was normal.

Investigations revealed:

Haemoglobin	102 g/L	(130 - 180)
White cell count	3.2×10^9/L	(4 - 11)
Platelets	212×10^9/L	(150 - 400)
ESR (Westergren)	145 mm/1st hour	(0 - 15)
Serum sodium	135 mmol/L	(137 - 144)
Serum potassium	3.4 mmol/L	(3.5 - 4.9)
Serum urea	3.4 mmol/L	(2.5 - 7.5)
Serum creatinine	120 umol/L	(60 - 110)
Serum creatine kinase	150 U/L	(24 - 170)
Serum albumin	21 g/L	(37 - 49)
Fasting plasma glucose	5.6 mmol/L	(3 - 6)
Serum cholesterol	6.7 mmol/L	(< 5.2)
Anti-Nuclear antibody	Positive	-
Rheumatoid factor	Positive	-
Extractable nuclear antigens	Negative	-

Urinalysis showed:

Protein	+++
Red cells	0
White cells	0
Gram stain	No organisms detected

What is the most likely cause for this patient's symptoms?

(Please select 1 option)

○ Guillain-Barré syndrome

○ Drug-induced myasthenic syndrome

○ Steroid myopathy

○ Polymyalgia rheumatica

○ Polymyositis

Submit Answer

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●	Drug-induced myasthenic syndrome **Correct**
○	Steroid myopathy
○	Polymyalgia rheumatica
○	Polymyositis

Key Learning Points

Neurology

- Drug induced myastheic syndromes can occur due to penacillamine toxicity.

Explanation

This patient presents with proximal limb weakness, ptosis and ocular paresis. In addition, she has deranged biochemistry with hypercholesterolaemia, hypoalbuminaemia and proteinuria.

This patient has probable penicillamine toxicity with nephrotic syndrome and myasthenic syndrome.

There is nothing in the history to suggest Guillain-Barré syndrome, which can present with proximal weakness.

Polymyalgia rheumatica presents with pain and stiffness normally affecting the shoulder girdle and is associated with an elevated erythrocyte sedimentation rate.

Polymyositis can occur as a result of penicillamine, but given the normal creatine kinase is less likely.

A steroid myopathy is a possibility in this patient, but it would be unusual to affect ocular musculature.

Question 51

Question 51 of 133

Core Questions

‹ ›

A 35-year-old man with epilepsy presents complaining of tunnel vision.

Which is the most likely drug responsible for this problem?

(Please select 1 option)

○	Levetiracetam
○	Sodium valproate
○	Carbamazepine
○	Topiramate
○	Vigabatrin

Submit Answer

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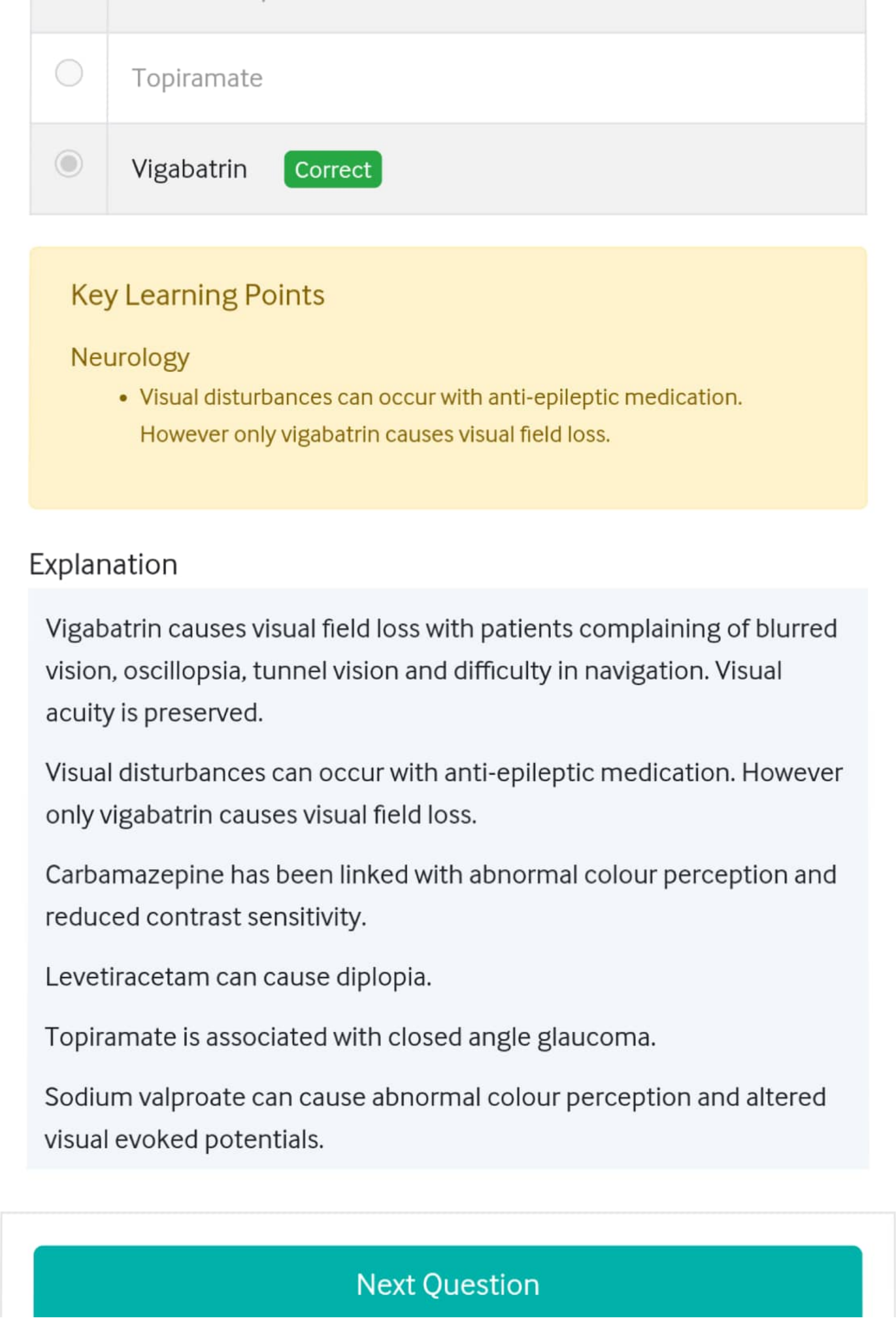


○	Topiramate
●	Vigabatrin Correct

Key Learning Points

Neurology

- Visual disturbances can occur with anti-epileptic medication. However only vigabatrin causes visual field loss.

Explanation

Vigabatrin causes visual field loss with patients complaining of blurred vision, oscillopsia, tunnel vision and difficulty in navigation. Visual acuity is preserved.

Visual disturbances can occur with anti-epileptic medication. However only vigabatrin causes visual field loss.

Carbamazepine has been linked with abnormal colour perception and reduced contrast sensitivity.

Levetiracetam can cause diplopia.

Topiramate is associated with closed angle glaucoma.

Sodium valproate can cause abnormal colour perception and altered visual evoked potentials.

Question 52 of 133

A 78-year-old man is admitted to hospital having developed abnormal movements of his left side. He has long-standing hypertension, well-controlled on atenolol, but has otherwise been healthy.

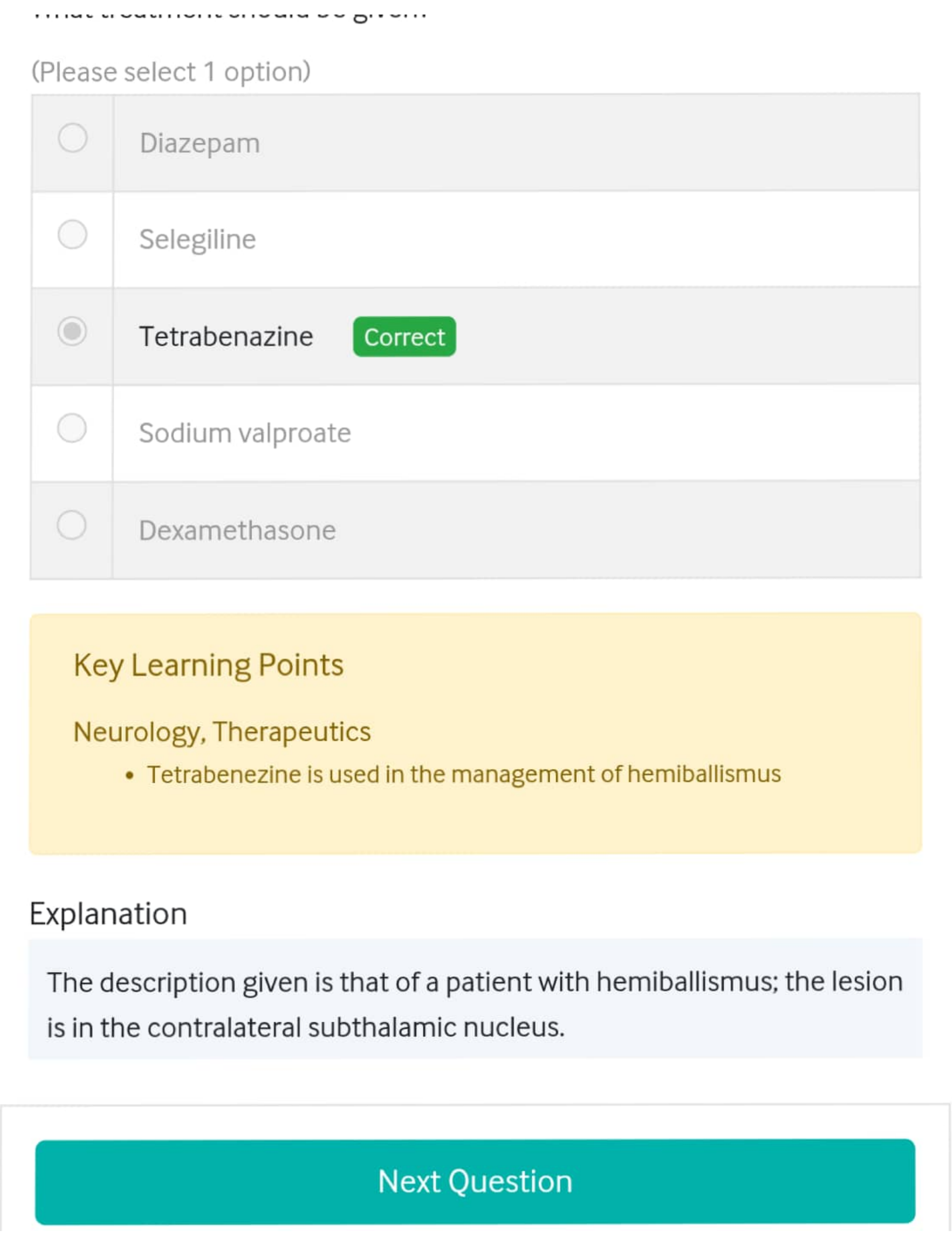
On examination of his central nervous system, he exhibits sudden, violent, involuntary flinging movements of his left arm.

What treatment should be given?

(Please select 1 option)

○	Diazepam
○	Selegiline
○	Tetrabenazine
○	Sodium valproate
○	Dexamethasone

Submit Answer



What treatment should be given?

(Please select 1 option)

○ Diazepam

○ Selegiline

● Tetrabenazine **Correct**

○ Sodium valproate

○ Dexamethasone

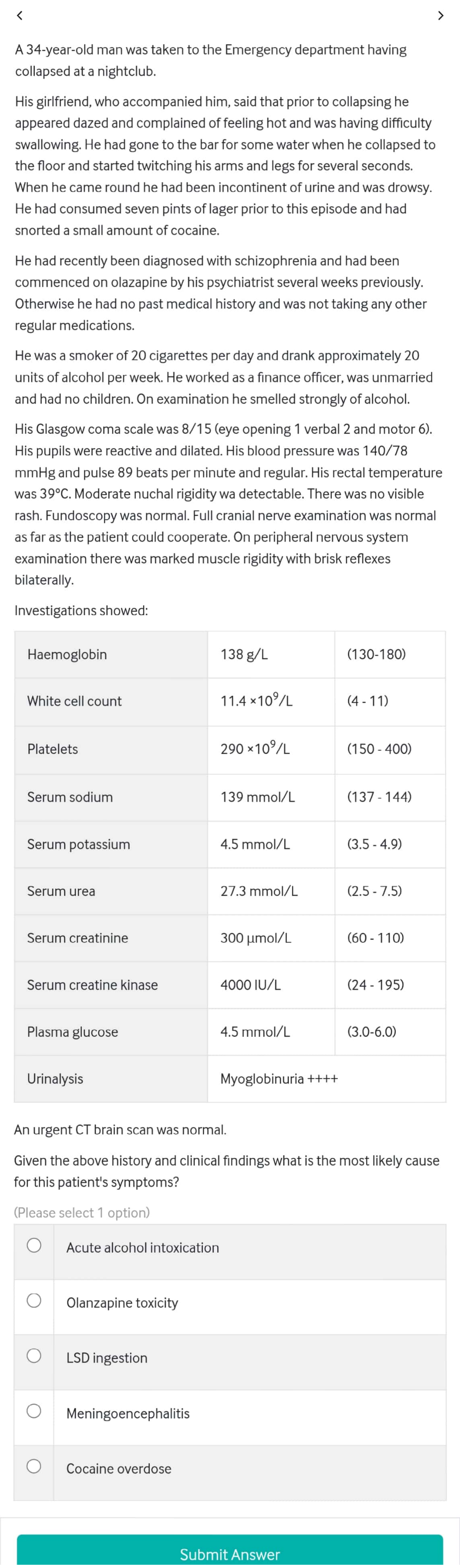
Key Learning Points

Neurology, Therapeutics

- Tetrabenezine is used in the management of hemiballismus

Explanation

The description given is that of a patient with hemiballismus; the lesion is in the contralateral subthalamic nucleus.



A 34-year-old man was taken to the Emergency department having collapsed at a nightclub.

His girlfriend, who accompanied him, said that prior to collapsing he appeared dazed and complained of feeling hot and was having difficulty swallowing. He had gone to the bar for some water when he collapsed to the floor and started twitching his arms and legs for several seconds. When he came round he had been incontinent of urine and was drowsy. He had consumed seven pints of lager prior to this episode and had snorted a small amount of cocaine.

He had recently been diagnosed with schizophrenia and had been commenced on olazapine by his psychiatrist several weeks previously. Otherwise he had no past medical history and was not taking any other regular medications.

He was a smoker of 20 cigarettes per day and drank approximately 20 units of alcohol per week. He worked as a finance officer, was unmarried and had no children. On examination he smelled strongly of alcohol.

His Glasgow coma scale was 8/15 (eye opening 1 verbal 2 and motor 6). His pupils were reactive and dilated. His blood pressure was 140/78 mmHg and pulse 89 beats per minute and regular. His rectal temperature was 39°C. Moderate nuchal rigidity wa detectable. There was no visible rash. Fundoscopy was normal. Full cranial nerve examination was normal as far as the patient could cooperate. On peripheral nervous system examination there was marked muscle rigidity with brisk reflexes bilaterally.

Investigations showed:

Haemoglobin	138 g/L	(130-180)
White cell count	11.4 $\times 10^9$/L	(4 - 11)
Platelets	290 $\times 10^9$/L	(150 - 400)
Serum sodium	139 mmol/L	(137 - 144)
Serum potassium	4.5 mmol/L	(3.5 - 4.9)
Serum urea	27.3 mmol/L	(2.5 - 7.5)
Serum creatinine	300 µmol/L	(60 - 110)
Serum creatine kinase	4000 IU/L	(24 - 195)
Plasma glucose	4.5 mmol/L	(3.0-6.0)
Urinalysis	Myoglobinuria ++++	

An urgent CT brain scan was normal.

Given the above history and clinical findings what is the most likely cause for this patient's symptoms?

(Please select 1 option)

○ Acute alcohol intoxication

○ Olanzapine toxicity

○ LSD ingestion

○ Meningoencephalitis

○ Cocaine overdose

○ Acute alcohol intoxication

○ Olanzapine toxicity **This is the correct answer**

○ LSD ingestion

○ Meningoencephalitis

● Cocaine overdose **Incorrect answer selected**

Key Learning Points

Neurology

- Neuroleptic malignant syndrome can be precipitated by Olanzapine.

Explanation

The most likely diagnosis in this patient is neuroleptic malignant syndrome as a result of olanzapine toxicity.

This patient presents with confusion, hyperthermia, muscle rigidity and prior history of autonomic dysfunction (dysphagia). He also has acute renal failure with myoglobinuria and markedly elevated creatine kinase.

Neuroleptic malignant syndrome is a serious side effect of neuroleptic therapy and is associated with high mortality rate.

Treatment consists of stopping the neuroleptic drug, and supporting measures including intravenous fluids, antipyretics and a cooling blanket.

Dopamine agonists, levadopa, amantadine and dantrolene have all been shown to be effective.

Recovery usually occurs within two to 14 days.

Question 54 of 133

< >

A 28-year-old shop worker is referred with a three month history of recurrent episodes of disorientation and confusion.

Her boyfriend has found her wandering around the house on several occasions, apparently with no idea of where she is or how she got there. Her mood has been very low, with frequent emotional outbursts, and she has considered leaving her job because of problems with working the computer and managing customers' queries. Her boyfriend feels her condition is significantly worsening.

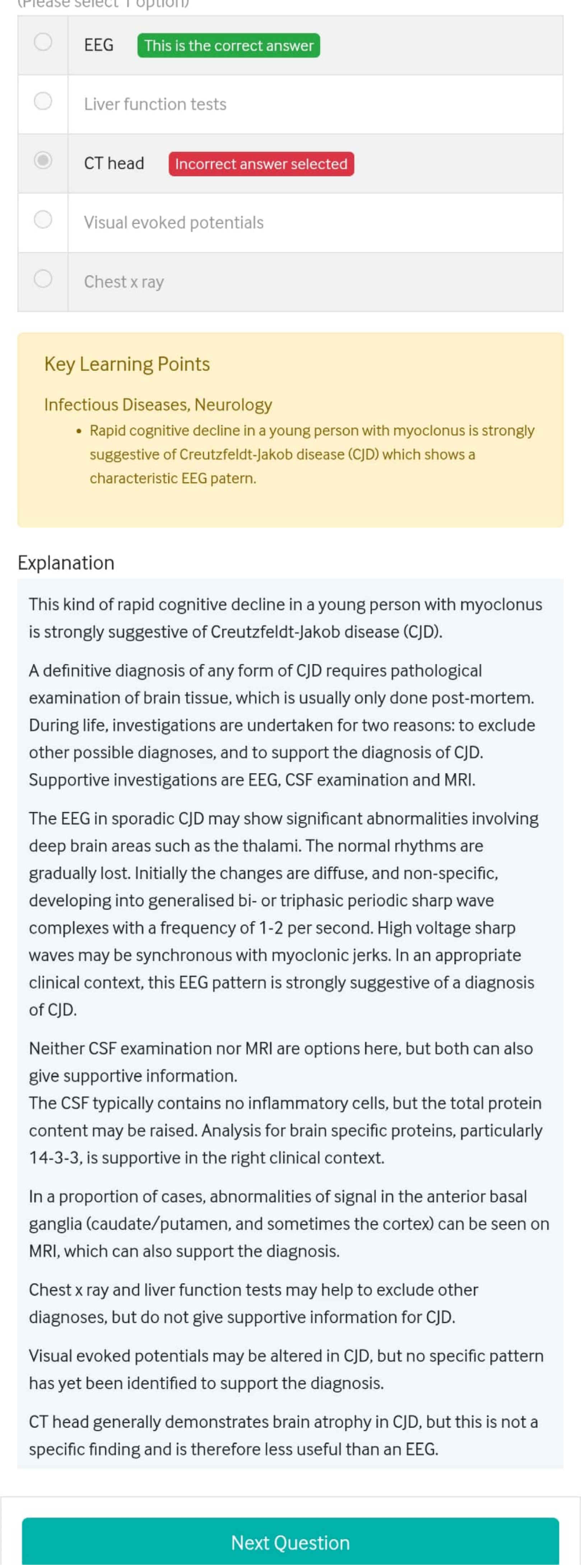
Physical examination is normal, apart from recurrent, asymetrical, jerks in all four limbs.

Which of the following investigations is likely to be most useful in reaching a diagnosis?

(Please select 1 option)

- ○ EEG
- ○ Liver function tests
- ○ CT head
- ○ Visual evoked potentials
- ○ Chest x ray

Submit Answer



(Please select 1 option)

○ EEG — This is the correct answer

○ Liver function tests

● CT head — Incorrect answer selected

○ Visual evoked potentials

○ Chest x ray

Key Learning Points

Infectious Diseases, Neurology

- Rapid cognitive decline in a young person with myoclonus is strongly suggestive of Creutzfeldt-Jakob disease (CJD) which shows a characteristic EEG patern.

Explanation

This kind of rapid cognitive decline in a young person with myoclonus is strongly suggestive of Creutzfeldt-Jakob disease (CJD).

A definitive diagnosis of any form of CJD requires pathological examination of brain tissue, which is usually only done post-mortem. During life, investigations are undertaken for two reasons: to exclude other possible diagnoses, and to support the diagnosis of CJD. Supportive investigations are EEG, CSF examination and MRI.

The EEG in sporadic CJD may show significant abnormalities involving deep brain areas such as the thalami. The normal rhythms are gradually lost. Initially the changes are diffuse, and non-specific, developing into generalised bi- or triphasic periodic sharp wave complexes with a frequency of 1-2 per second. High voltage sharp waves may be synchronous with myoclonic jerks. In an appropriate clinical context, this EEG pattern is strongly suggestive of a diagnosis of CJD.

Neither CSF examination nor MRI are options here, but both can also give supportive information.
The CSF typically contains no inflammatory cells, but the total protein content may be raised. Analysis for brain specific proteins, particularly 14-3-3, is supportive in the right clinical context.

In a proportion of cases, abnormalities of signal in the anterior basal ganglia (caudate/putamen, and sometimes the cortex) can be seen on MRI, which can also support the diagnosis.

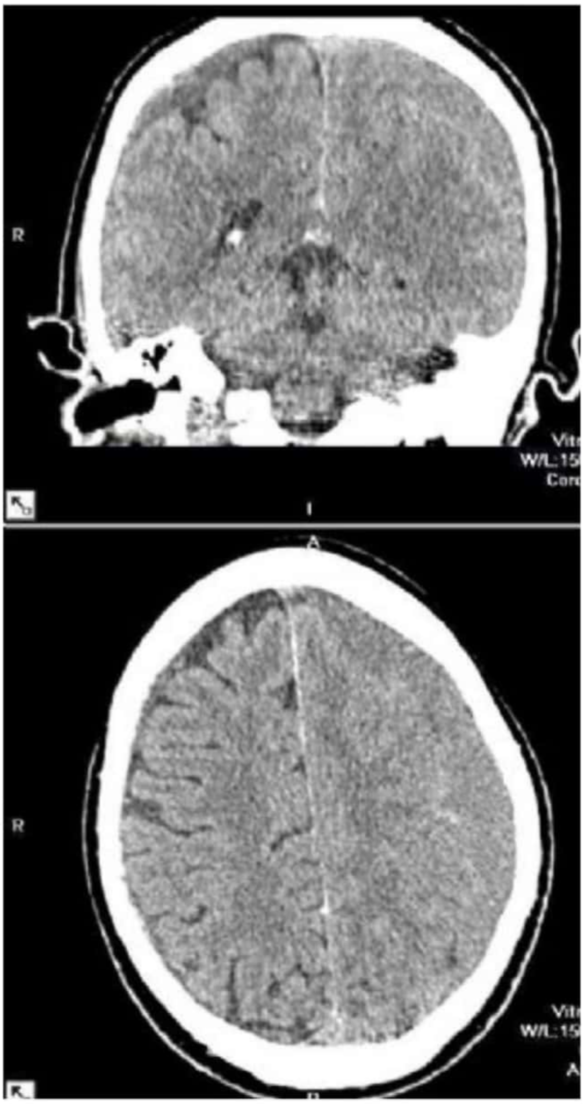
Chest x ray and liver function tests may help to exclude other diagnoses, but do not give supportive information for CJD.

Visual evoked potentials may be altered in CJD, but no specific pattern has yet been identified to support the diagnosis.

CT head generally demonstrates brain atrophy in CJD, but this is not a specific finding and is therefore less useful than an EEG.

A 70-year-old male is admitted to hospital after a fall in his nursing home. The nursing staff note that he is a little subdued and he is generally weak.

A CT scan is performed.



What is the diagnosis?

(Please select 1 option)

○ Subacute subdural haematoma

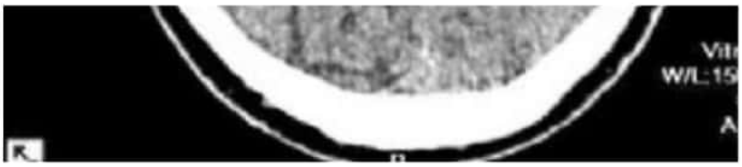
○ Cerebral infarct

○ Acute subarachnoid haemorrhage

○ Chronic subdural haematoma

○ Cerebral haemorrhage

Submit Answer



What is the diagnosis?

(Please select 1 option)

○ Subacute subdural haematoma **This is the correct answer**

○ Cerebral infarct

○ Acute subarachnoid haemorrhage

● Chronic subdural haematoma **Incorrect answer selected**

○ Cerebral haemorrhage

Key Learning Points

Neurology, Radiology

- Elderly and falls cay result in sudural haematoma, symptoms may be present subacutely

Explanation

There is a large left subdural haematoma.

The haematoma is isodense with the adjacent brain, which is compressed with complete loss of the sulci (the sulci are clearly visible on the right side).

There are no areas of high attenuation (white) blood to suggest an acute haemorrhage; equally, a chronic subdural would cause even lower attenuation blood (more black).

Histories that are as vague as this could easily be associated with an acute, subacute, or chronic subdural.

A 52-year-old woman was admitted to the Emergency department having developed severe back pain.

Within minutes she had developed weakness and paraesthesia affecting both her legs. She had been unable to urinate and the weakness in her legs had subsequently worsened. She had been well prior to her symptoms and had recently come back from a holiday in Tenerife.

She had a past medical history of diabetes for which she takes basal bolus insulin, hypertension treated with atenolol and hypercholesterolaemia for which she took simvastatin.

She did not smoke, but drank 15 units of alcohol per week. She was married with two children, one of whom had factor V Leiden mutation.

On examination she appeared distressed suffering from severe back pain. Her blood pressure was 156/90 mmHg, pulse was 92/minute and regular, temperature was 37.1°C and heart sounds were normal. Chest was clear and abdominal examination revealed a palpable bladder.

On neurological examination, she was alert and orientated. There were no abnormalities within the cranial nerves and fundoscopy revealed some cotton wool spots and flame haemorrhages. Examination of the upper limb revealed normal tone, power, reflexes and sensation. Examination of the lower limb revealed flaccid tone bilaterally, paraplegia, with areflexia and absent plantar responses. Sensory examination revealed impaired pinprick and temperature appreciation extending to a level just above the umbilicus. Light touch and vibration sense were intact. Cerebellar function was not tested.

A lumbar puncture was performed and revealed:

CSF opening pressure	12 cmH_2O	(50-180)
CSF white cell count	4 cells per ml	(<5)
CSF red cell count	3 cells per ml	(<5)
CSF protein	0.45 g/L	(0.15-0.45)
CSF glucose	4.2 mmol/L	(3.3-4.4)
CSF culture	Negative	-

The chest x ray showed a mediastinal width of 6 cm, but nothing else of note. A twelve-lead ECG was normal.

Given the above clinical account and investigations so far, what is the most likely diagnosis?

(Please select 1 option)

○ Guillain-Barré syndrome

○ Transverse myelitis

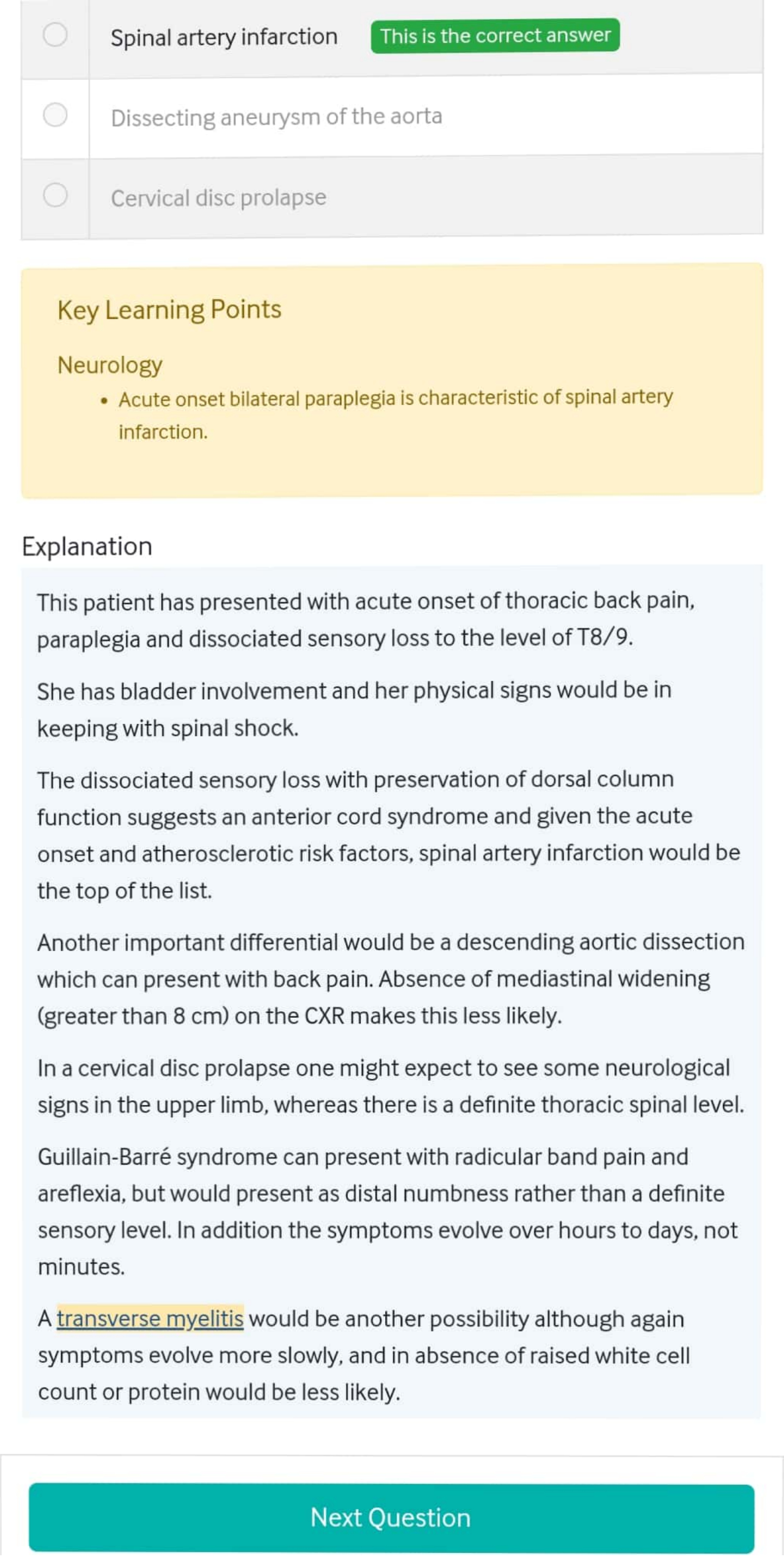
○ Spinal artery infarction

○ Dissecting aneurysm of the aorta

○ Cervical disc prolapse

Submit Answer

Skip Question



○ Spinal artery infarction **This is the correct answer**

○ Dissecting aneurysm of the aorta

○ Cervical disc prolapse

Key Learning Points

Neurology

- Acute onset bilateral paraplegia is characteristic of spinal artery infarction.

Explanation

This patient has presented with acute onset of thoracic back pain, paraplegia and dissociated sensory loss to the level of T8/9.

She has bladder involvement and her physical signs would be in keeping with spinal shock.

The dissociated sensory loss with preservation of dorsal column function suggests an anterior cord syndrome and given the acute onset and atherosclerotic risk factors, spinal artery infarction would be the top of the list.

Another important differential would be a descending aortic dissection which can present with back pain. Absence of mediastinal widening (greater than 8 cm) on the CXR makes this less likely.

In a cervical disc prolapse one might expect to see some neurological signs in the upper limb, whereas there is a definite thoracic spinal level.

Guillain-Barré syndrome can present with radicular band pain and areflexia, but would present as distal numbness rather than a definite sensory level. In addition the symptoms evolve over hours to days, not minutes.

A transverse myelitis would be another possibility although again symptoms evolve more slowly, and in absence of raised white cell count or protein would be less likely.

A 32-year-old woman presented to the Emergency department with a sudden onset weakness and numbness affecting the left side of the face, arm and leg. She had been out shopping with a friend at the time when she had developed a typical migraine attack, shortly after the weakness and numbness came on. There was no disturbance of consciousness, vision or speech.

She had a past medical history of troublesome migraines associated with aura and had a previous episode of right arm weakness six months ago, which had lasted 20 minutes before she recovered. She took regular pizotifen 1.5 mg and Maxalt melt and was also on the oral contraceptive pill. There was a family history of migraine and strokes and her mother died in her 50s with dementia. She was a smoker of 10 cigarettes per day and did not drink any alcohol.

On examination her blood pressure was 135/75 mmHg, pulse was 65/min and regular and heart sounds were normal. On cranial nerve examination there was left facial asymmetry and weakness with reduced sensation over the left face extending to the vertex. Pupils and fundoscopy were all normal. Peripheral nervous system examination revealed mild left hemiparesis of 4/5 with hyper-reflexia and left extensor plantar response. There was numbness to all modalities over the left arm and leg.

A MRI scan of the brain showed bilateral, multifocal, T2/FLAIR hyperintensities in the deep white matter.

A lumbar puncture was performed and yielded the following data:

Opening pressure	8 cmH_2O	(6-18)
CSF protein	0.35 g/L	(0.15-0.45)
CSF white cell count	3 cells per mL	(≤5)
CSF red cell count	2 cells per mL	(≤5)
CSF lactate	1.1 mmol/L	(1-2)
CSF oligoclonal bands	Negative	

Given the above history and clinical findings, how should this patient be further managed?

(Please select 1 option)

○ Increase the pizotifen to 3 mg/day

○ Arrange commencement of beta interferon

○ Arrange a cerebral angiogram

○ Commence on intravenous methylprednisolone

○ Stop the oral contraceptive pill

○ Arrange a cerebral angiogram

○ Commence on intravenous methylprednisolone

○ Stop the oral contraceptive pill This is the correct answer

Key Learning Points

Neurology

- OCP can cause stroke like episodes, in patients suffering with migraine.

Explanation

This patient has a background of migraine and appears to have had two stroke-like episodes.

There is also a family history of migraine, strokes and dementia.

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) should be considered in the diagnosis.

DNA testing for the notch-3 gene mutation confirms the diagnosis.

In addition, the oral contraceptive pill should be stopped, given its association with stroke in migraineurs.

Next Question

A 48-year-old woman presented to her GP with a left wrist drop.

She was unsure how it started but had noticed weakness of her wrist and fingers for over a week. In addition, she also complained of numbness over the posterior aspect of the left forearm and dorsum of the left hand and had recently had some neck pain.

She had a history of depression and alcohol abuse and took regular fluoxetine 20 mg/day. Otherwise, she was fit and well and had no family history of medical problems.

On examination of the upper limb, there was no apparent wasting or fasciculation. On examination of tone, there was slight reduction at the left elbow and wrist. The biceps, triceps and supinator reflexes were also depressed in the left arm and normal in the right. On examination of power, there was weakness of left elbow extension, flexion of the elbow with the forearm midway between pronation and supination, supination of the forearm, extension of the wrist and fingers and extension and abduction of the thumb in the plane of the palm. Shoulder abduction appeared normal. Sensation was impaired over the posterior forearm and also the dorsum of the hand.

An MRI scan of the cervical spine showed mild degenerative disc disease affecting C5-C7.

Where will the lesion be on nerve conduction studies?

(Please select 1 option)

○ Motor neurone disease affecting C7 anterior horn cells

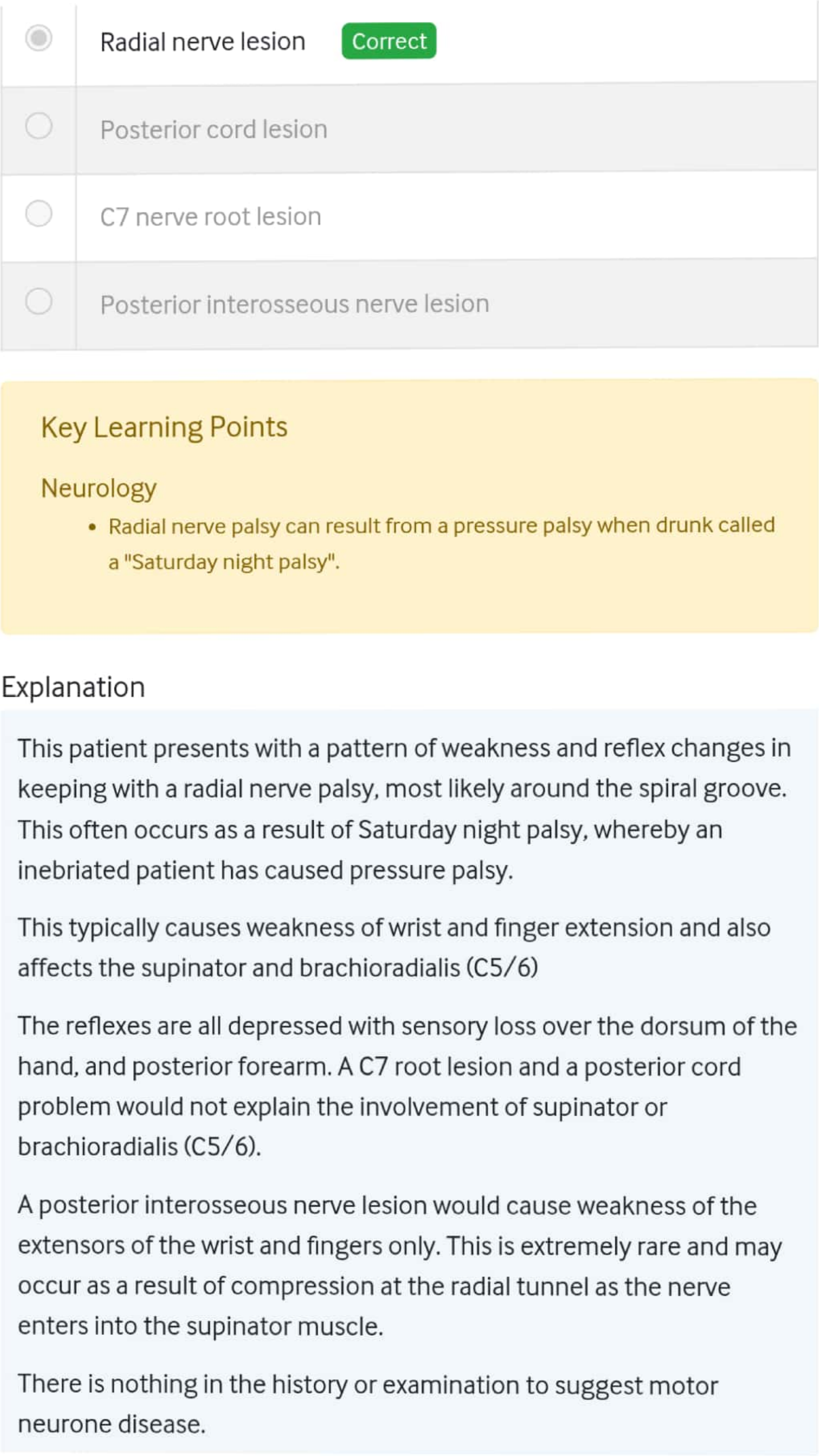
○ Radial nerve lesion

○ Posterior cord lesion

○ C7 nerve root lesion

○ Posterior interosseous nerve lesion

Submit Answer



● Radial nerve lesion **Correct**

○ Posterior cord lesion

○ C7 nerve root lesion

○ Posterior interosseous nerve lesion

Key Learning Points

Neurology

- Radial nerve palsy can result from a pressure palsy when drunk called a "Saturday night palsy".

Explanation

This patient presents with a pattern of weakness and reflex changes in keeping with a radial nerve palsy, most likely around the spiral groove. This often occurs as a result of Saturday night palsy, whereby an inebriated patient has caused pressure palsy.

This typically causes weakness of wrist and finger extension and also affects the supinator and brachioradialis (C5/6)

The reflexes are all depressed with sensory loss over the dorsum of the hand, and posterior forearm. A C7 root lesion and a posterior cord problem would not explain the involvement of supinator or brachioradialis (C5/6).

A posterior interosseous nerve lesion would cause weakness of the extensors of the wrist and fingers only. This is extremely rare and may occur as a result of compression at the radial tunnel as the nerve enters into the supinator muscle.

There is nothing in the history or examination to suggest motor neurone disease.

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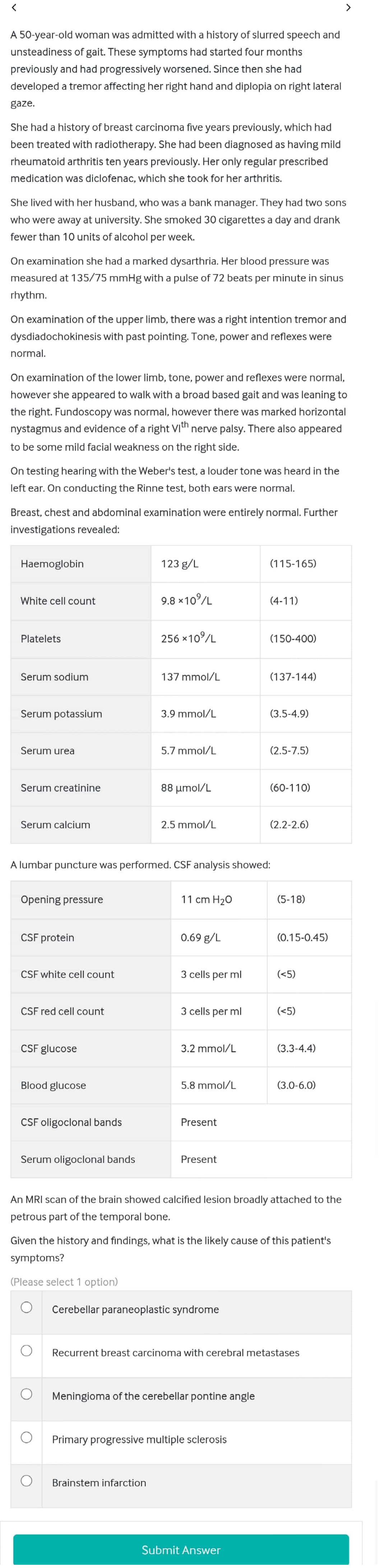


A 50-year-old woman was admitted with a history of slurred speech and unsteadiness of gait. These symptoms had started four months previously and had progressively worsened. Since then she had developed a tremor affecting her right hand and diplopia on right lateral gaze.

She had a history of breast carcinoma five years previously, which had been treated with radiotherapy. She had been diagnosed as having mild rheumatoid arthritis ten years previously. Her only regular prescribed medication was diclofenac, which she took for her arthritis.

She lived with her husband, who was a bank manager. They had two sons who were away at university. She smoked 30 cigarettes a day and drank fewer than 10 units of alcohol per week.

On examination she had a marked dysarthria. Her blood pressure was measured at 135/75 mmHg with a pulse of 72 beats per minute in sinus rhythm.

On examination of the upper limb, there was a right intention tremor and dysdiadochokinesis with past pointing. Tone, power and reflexes were normal.

On examination of the lower limb, tone, power and reflexes were normal, however she appeared to walk with a broad based gait and was leaning to the right. Fundoscopy was normal, however there was marked horizontal nystagmus and evidence of a right VIth nerve palsy. There also appeared to be some mild facial weakness on the right side.

On testing hearing with the Weber's test, a louder tone was heard in the left ear. On conducting the Rinne test, both ears were normal.

Breast, chest and abdominal examination were entirely normal. Further investigations revealed:

Haemoglobin	123 g/L	(115-165)
White cell count	9.8 $\times 10^9$/L	(4-11)
Platelets	256 $\times 10^9$/L	(150-400)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	3.9 mmol/L	(3.5-4.9)
Serum urea	5.7 mmol/L	(2.5-7.5)
Serum creatinine	88 µmol/L	(60-110)
Serum calcium	2.5 mmol/L	(2.2-2.6)

A lumbar puncture was performed. CSF analysis showed:

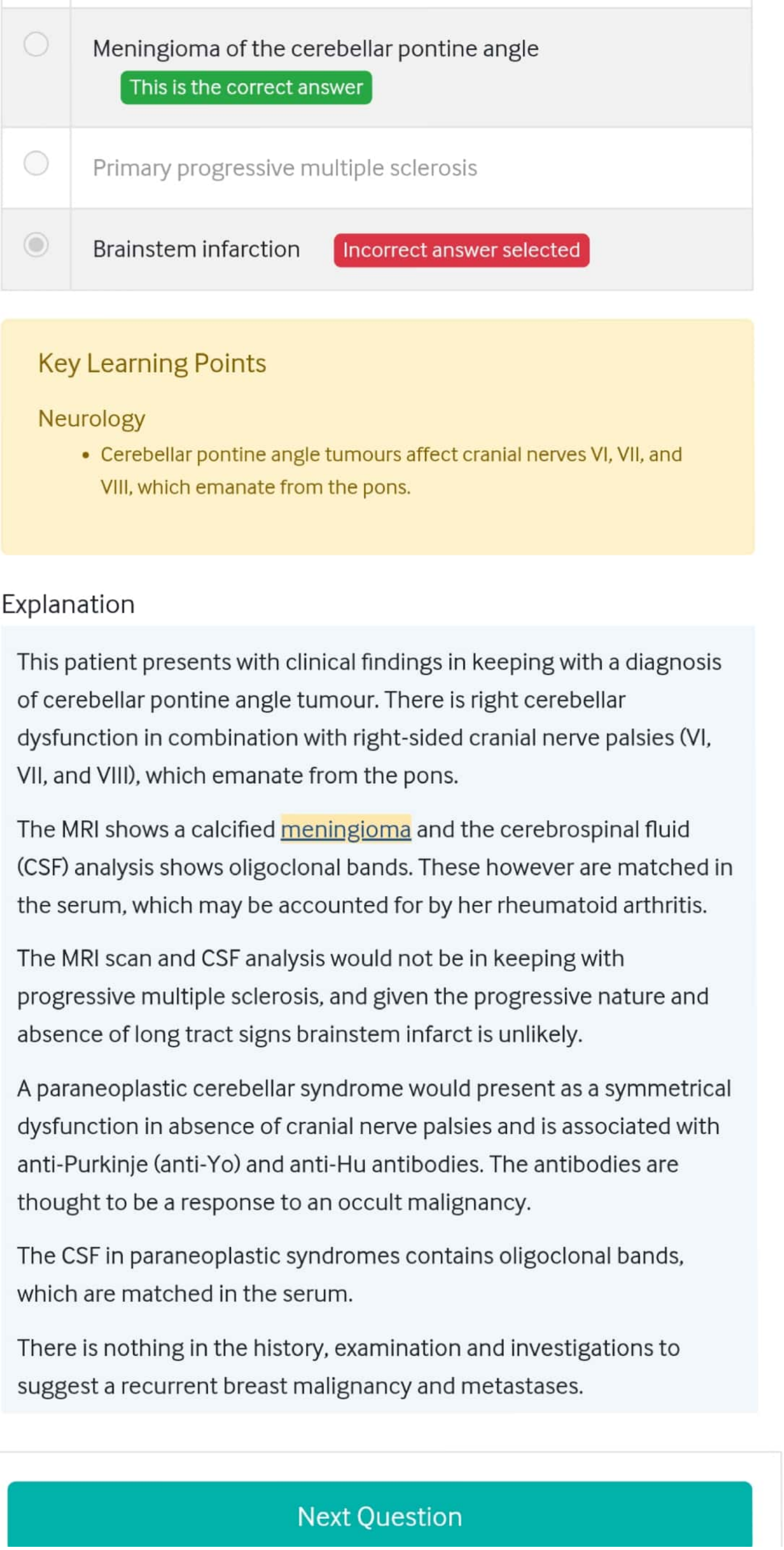
Opening pressure	11 cm H_2O	(5-18)
CSF protein	0.69 g/L	(0.15-0.45)
CSF white cell count	3 cells per ml	(<5)
CSF red cell count	3 cells per ml	(<5)
CSF glucose	3.2 mmol/L	(3.3-4.4)
Blood glucose	5.8 mmol/L	(3.0-6.0)
CSF oligoclonal bands	Present	
Serum oligoclonal bands	Present	

An MRI scan of the brain showed calcified lesion broadly attached to the petrous part of the temporal bone.

Given the history and findings, what is the likely cause of this patient's symptoms?

(Please select 1 option)

- ○ Cerebellar paraneoplastic syndrome
- ○ Recurrent breast carcinoma with cerebral metastases
- ○ Meningioma of the cerebellar pontine angle
- ○ Primary progressive multiple sclerosis
- ○ Brainstem infarction



○ Meningioma of the cerebellar pontine angle

This is the correct answer

○ Primary progressive multiple sclerosis

● Brainstem infarction Incorrect answer selected

Key Learning Points

Neurology

- Cerebellar pontine angle tumours affect cranial nerves VI, VII, and VIII, which emanate from the pons.

Explanation

This patient presents with clinical findings in keeping with a diagnosis of cerebellar pontine angle tumour. There is right cerebellar dysfunction in combination with right-sided cranial nerve palsies (VI, VII, and VIII), which emanate from the pons.

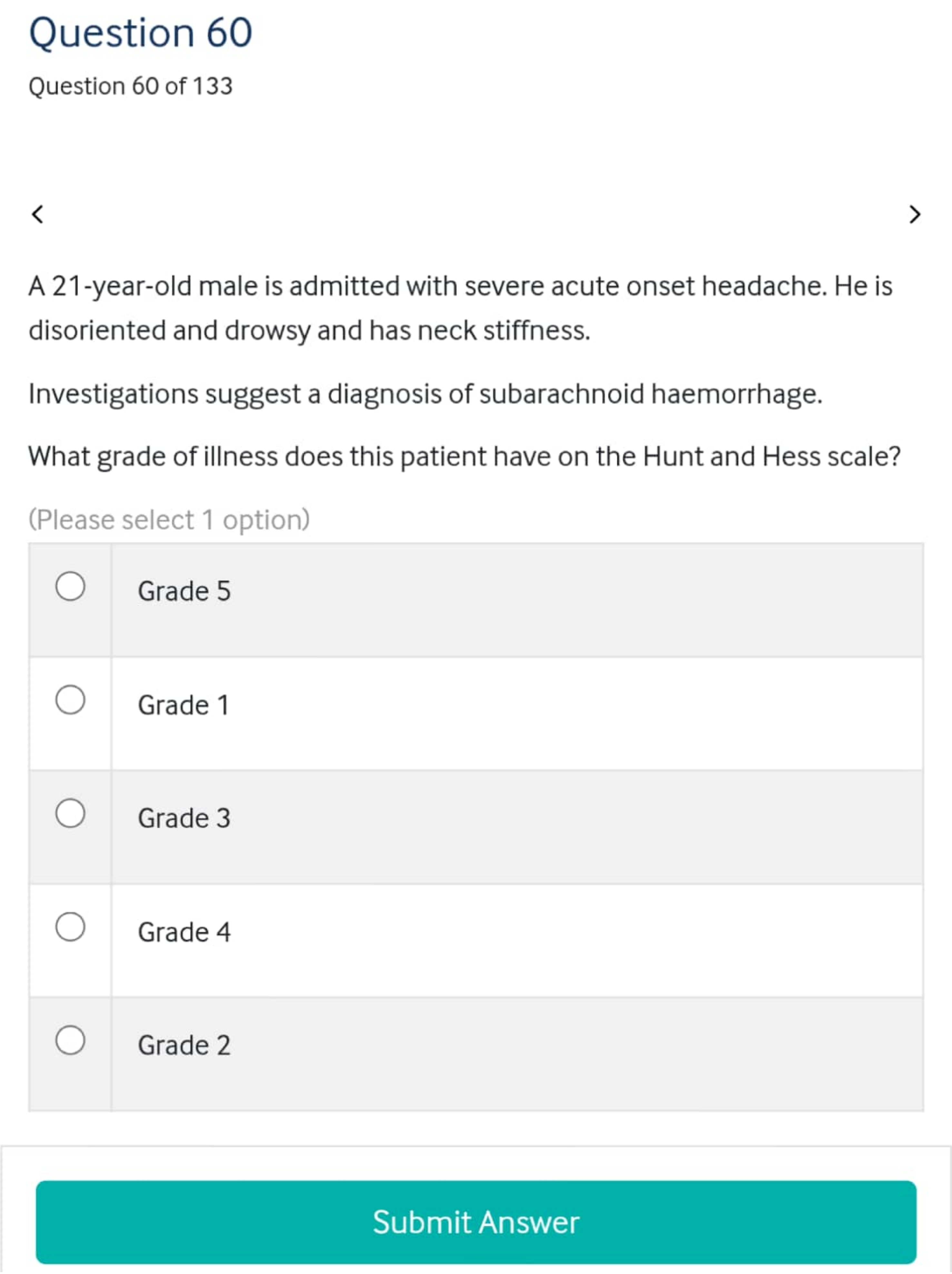
The MRI shows a calcified meningioma and the cerebrospinal fluid (CSF) analysis shows oligoclonal bands. These however are matched in the serum, which may be accounted for by her rheumatoid arthritis.

The MRI scan and CSF analysis would not be in keeping with progressive multiple sclerosis, and given the progressive nature and absence of long tract signs brainstem infarct is unlikely.

A paraneoplastic cerebellar syndrome would present as a symmetrical dysfunction in absence of cranial nerve palsies and is associated with anti-Purkinje (anti-Yo) and anti-Hu antibodies. The antibodies are thought to be a response to an occult malignancy.

The CSF in paraneoplastic syndromes contains oligoclonal bands, which are matched in the serum.

There is nothing in the history, examination and investigations to suggest a recurrent breast malignancy and metastases.



Question 60

Question 60 of 133

A 21-year-old male is admitted with severe acute onset headache. He is disoriented and drowsy and has neck stiffness.

Investigations suggest a diagnosis of subarachnoid haemorrhage.

What grade of illness does this patient have on the Hunt and Hess scale?

(Please select 1 option)

- ○ Grade 5
- ○ Grade 1
- ○ Grade 3
- ○ Grade 4
- ○ Grade 2

○ Grade 1

● Grade 3 Correct

○ Grade 4

○ Grade 2

Key Learning Points

Neurology

- The Hunt and Hess scale grades subarachnoid haemorrhage

Explanation

This gentleman's history is suggestive of subarachnoid haemorrhage (SAH).

The Hunt and Hess scale grades SAH thus:

1. Asymptomatic or minimal headache & slight neck stiffness
2. Moderate or severe headache with neck stiffness, but no neurological deficit other than cranial nerve palsy
3. Drowsiness with confusion or mild focal neurology
4. Stupor with moderate to severe hemiparesis or mild decerebrate rigidity
5. Deeply comatose with severe decerebrate rigidity.

Severity and mortality increase with grade.

Reference:

Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. *J Neurosurg*. 1968;28:14-20.

Next Question

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A 40-year-old woman was admitted to the Emergency department with generalised tonic-clonic seizures.

Her flatmate, who accompanied her to hospital, reported that the patient had complained of headaches for several weeks. In the past week she had been vomiting several times per day and had become slightly confused and disorientated.

She had worked as a tour guide for twenty years for a package holiday company and had recently returned from Australia, where she had been working for six months. There was no history of illness while she had been overseas, but had noticed slight weight loss, which she had attributed to increased sporting activity.

She was a smoker of 20 cigarettes per day and drank 21 units of alcohol per week. There was no other past medical history of note and she was not taking any regular medication.

On examination she appeared confused with a Glasgow coma scale of 13/15 (motor 6 eyes 4 vocal 13). Her blood pressure was 135/67 mmHg, pulse was 78 beats per minute and regular with a temperature of 36.7°C. There was mild neck stiffness and photophobia. She had enlarged lymph nodes in the neck, axilla and in the left inguinal region.

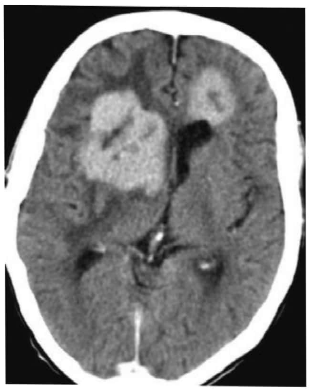
There were two macular brown lesions on the left leg and over the abdomen. Fundoscopy was normal and pupillary responses were intact. The remainder of the cranial nerve examination was normal.

On examination of the peripheral nervous system, tone and power were normal. Reflexes were bilaterally brisk and both plantar responses were extensor. Full examination of the chest and abdomen were normal.

Investigations revealed:

Haemoglobin	136 g/L	(130 - 180)
White cell count	11 $\times 10^9$/L	(4 - 11)
Platelets	356 $\times 10^9$/L	(150 - 400)
C reactive protein	8 mg/L	(<10)
Serum sodium	136 mmol/L	(137 - 144)
Serum potassium	3.8 mmol/L	(3.5 - 4.9)
Serum urea	7.8 mmol/L	(2.5 - 7.5)
Serum creatinine	110 µmol/L	(60 - 110)
Serum calcium	2.3 mmol/L	(2.2-2.6)

A CT scan of the brain was performed. A non-contrasted section from this study is shown below:



Given the above history and investigations so far, what is the likely diagnosis in this patient?

(Please select 1 option)

○	Systemic lymphoma
○	Melanoma metastases
○	Tuberous sclerosis
○	Arteriovenous malformation
○	Meningioma

Submit Answer

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● Melanoma metastases **Correct**

○ Tuberous sclerosis

○ Arteriovenous malformation

○ Meningioma

Key Learning Points

Neurology, Oncology, Photographic, Radiology

- Melanoma commonly spreads to the brain and lesions can cavitate causing neurological decline.

Explanation

This patient has melanoma metastases that have cavitated.

Clues to this diagnosis include high-risk occupation for sun exposure and brown macular lesions over the trunk and leg (common places for superficial spreading melanomas).

The imaging findings are not in keeping with an arteriovenous malformation, although this diagnosis would need to be considered in young patients presenting with headache and seizures.

The lesions are intracerebral and not confined to the meninges. Primary lymphoma or systemic lymphoma with leptomeningeal spread would need to be considered in this patient, given the history.

It is quite unusual for systemic lymphoma to metastasise to the brain.

Primary lymphoma is associated with immunodeficiency and radiological findings are of dense homogenous enhancing masses.

The CT findings are not consistent with tubers found in tuberous sclerosis.

A 60-year-old right-handed man was admitted to the Emergency department with left-sided face and arm weakness and numbness that started approximately 45 minutes previously.

He had no associated leg weakness, swallowing or speech impairment, but had complained of headache at the time. He had a history of treated hypertension and was taking bendroflumethiazide. Otherwise he was fit and well, was a non-smoker and did not drink any alcohol.

On examination there was evidence of left facial asymmetry. His blood pressure was 138/65 mmHg, pulse was 78/min and irregular and heart sounds were normal. He had no carotid bruits or tender temporal arteries. On cranial nerve examination there was left upper motor neurone facial weakness and numbness, but the rest of the nerves including fundoscopy were intact.

On peripheral nerve examination there was pyramidal weakness of the left arm, with brisk jerks and positive Hoffmann's sign. There was reduced sensation over the entire limb. The lower limb appeared intact.

His overall National Institute of Health Stroke Scale (NIHSS) was 9/42.

A CT scan was performed within an hour of admission.

Investigations revealed:

Haemoglobin	134 g/L	(130-180)
White cell count	6.7 ×10^9/L	(4-11)
Platelets	234 ×10^9/L	(150-400)
Serum sodium	142 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	4.6 mmol/L	(2.5-7.5)
Serum creatinine	88 mol/L	(60-110)
Fasting plasma glucose	4.6 mmol/L	(3-6)
Serum cholesterol	3.6 mmol/L	(<5.2)
International Normalised Ratio	0.98	(<1.4)

A CT scan of the brain was normal. The electrocardiogram showed atrial fibrillation with a ventricular rate of 78 beats per minute.

Given the clinical history and the results of his investigations, how should this patient be ideally managed?

(Please select 1 option)

○ Commence on intravenous tissue plasminogen activator (t-PA)

○ Commence on warfarin

○ Commence on warfarin and load with intravenous digoxin

○ Commence on aspirin 300 mg and transfer to a stroke ward

○ Commence on aspirin 300 mg and switch bendroflumethiazide to perindopril

Submit Answer

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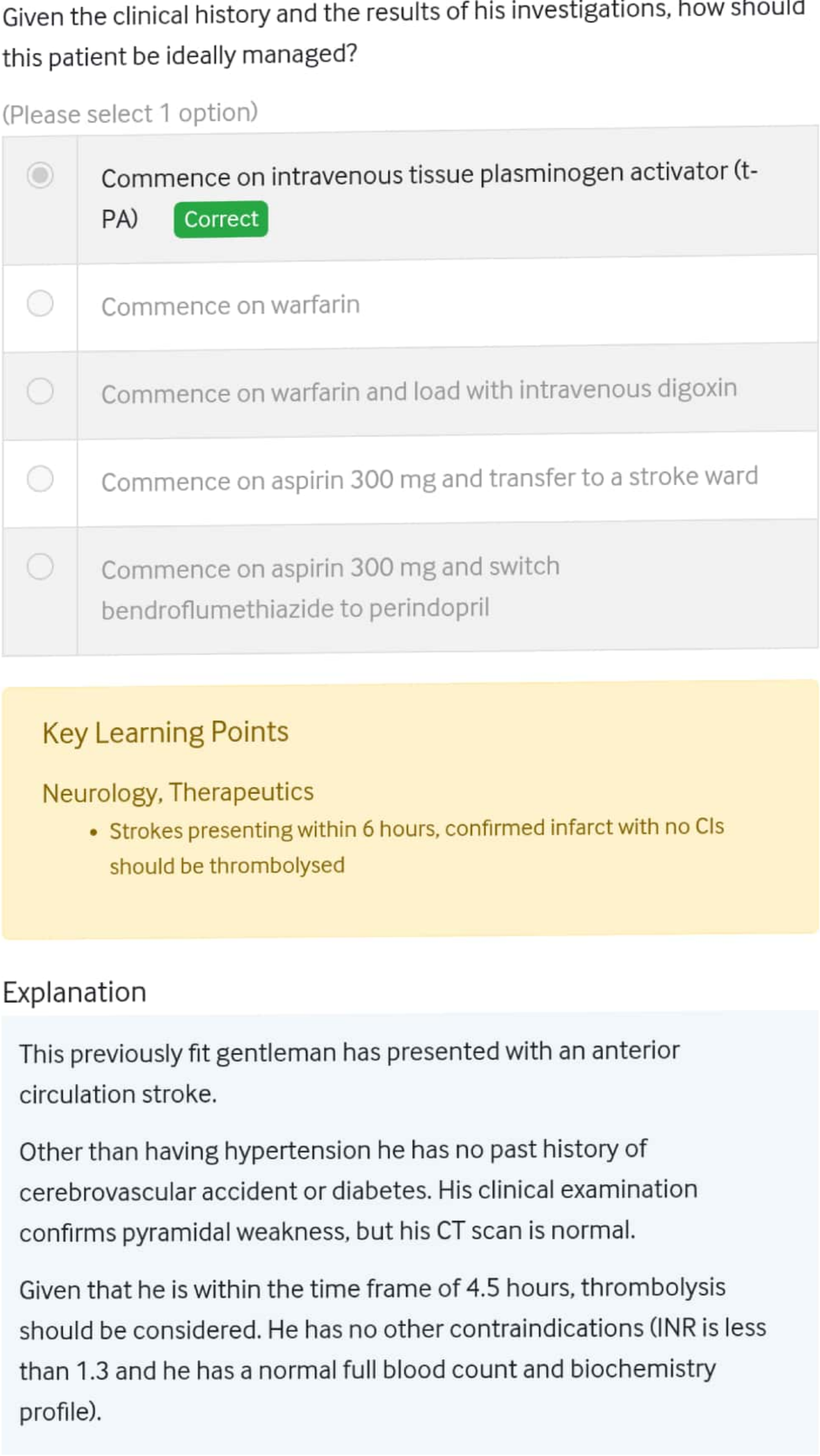


Given the clinical history and the results of his investigations, how should this patient be ideally managed?

(Please select 1 option)

- ● Commence on intravenous tissue plasminogen activator (t-PA) **Correct**
- ○ Commence on warfarin
- ○ Commence on warfarin and load with intravenous digoxin
- ○ Commence on aspirin 300 mg and transfer to a stroke ward
- ○ Commence on aspirin 300 mg and switch bendroflumethiazide to perindopril

Key Learning Points

Neurology, Therapeutics

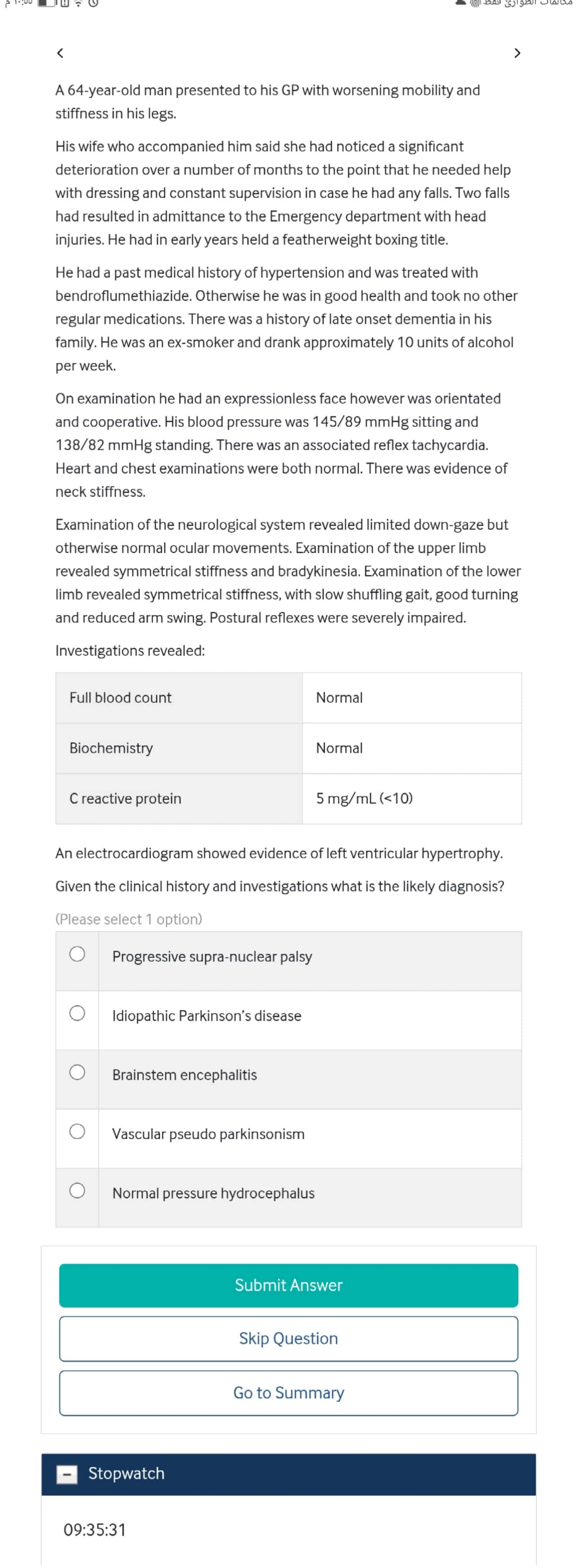
- Strokes presenting within 6 hours, confirmed infarct with no CIs should be thrombolysed

Explanation

This previously fit gentleman has presented with an anterior circulation stroke.

Other than having hypertension he has no past history of cerebrovascular accident or diabetes. His clinical examination confirms pyramidal weakness, but his CT scan is normal.

Given that he is within the time frame of 4.5 hours, thrombolysis should be considered. He has no other contraindications (INR is less than 1.3 and he has a normal full blood count and biochemistry profile).



A 64-year-old man presented to his GP with worsening mobility and stiffness in his legs.

His wife who accompanied him said she had noticed a significant deterioration over a number of months to the point that he needed help with dressing and constant supervision in case he had any falls. Two falls had resulted in admittance to the Emergency department with head injuries. He had in early years held a featherweight boxing title.

He had a past medical history of hypertension and was treated with bendroflumethiazide. Otherwise he was in good health and took no other regular medications. There was a history of late onset dementia in his family. He was an ex-smoker and drank approximately 10 units of alcohol per week.

On examination he had an expressionless face however was orientated and cooperative. His blood pressure was 145/89 mmHg sitting and 138/82 mmHg standing. There was an associated reflex tachycardia. Heart and chest examinations were both normal. There was evidence of neck stiffness.

Examination of the neurological system revealed limited down-gaze but otherwise normal ocular movements. Examination of the upper limb revealed symmetrical stiffness and bradykinesia. Examination of the lower limb revealed symmetrical stiffness, with slow shuffling gait, good turning and reduced arm swing. Postural reflexes were severely impaired.

Investigations revealed:

Full blood count	Normal
Biochemistry	Normal
C reactive protein	5 mg/mL (<10)

An electrocardiogram showed evidence of left ventricular hypertrophy.

Given the clinical history and investigations what is the likely diagnosis?

(Please select 1 option)

○ Progressive supra-nuclear palsy

○ Idiopathic Parkinson's disease

○ Brainstem encephalitis

○ Vascular pseudo parkinsonism

○ Normal pressure hydrocephalus

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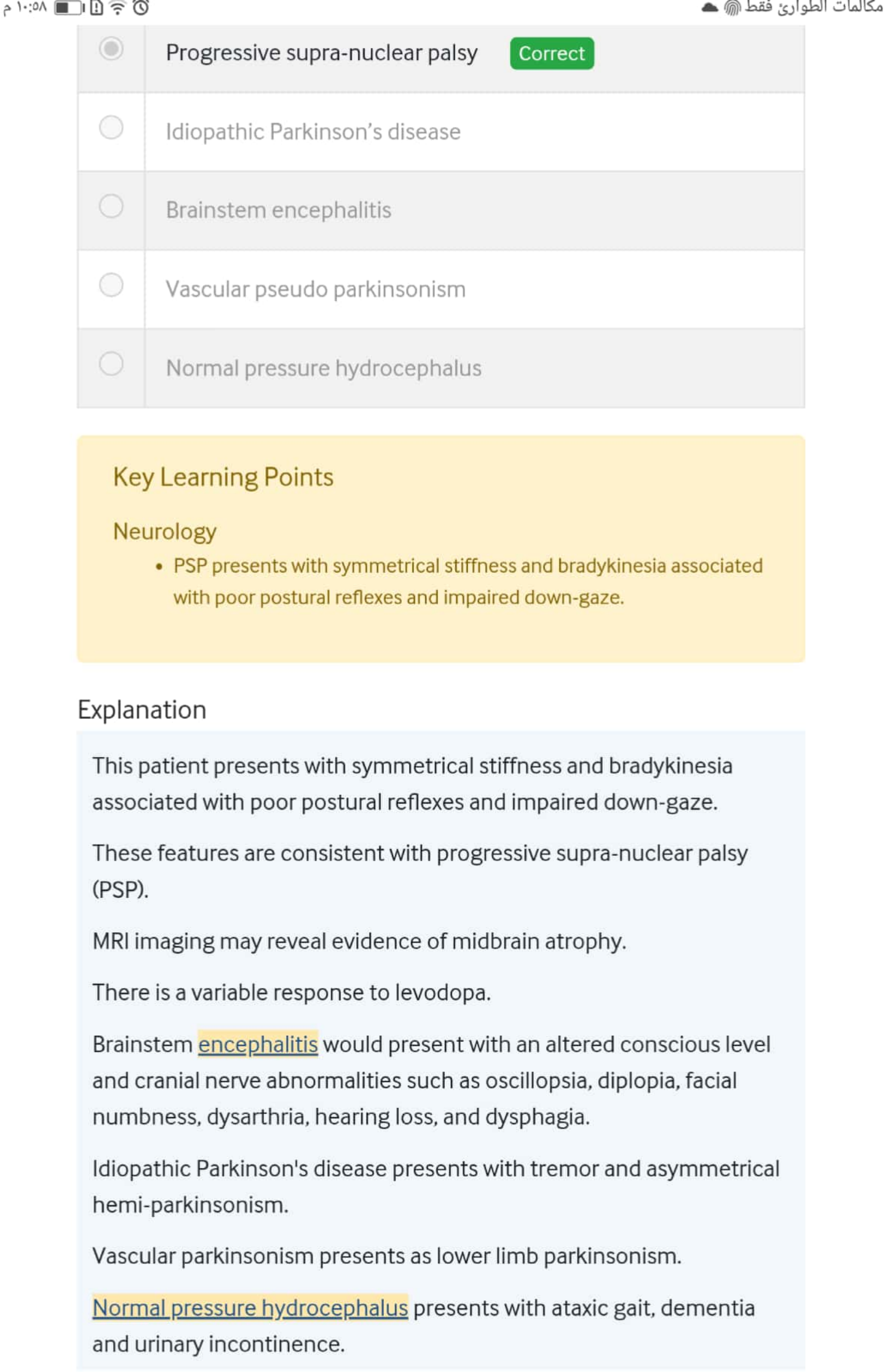


● Progressive supra-nuclear palsy

○ Idiopathic Parkinson's disease

○ Brainstem encephalitis

○ Vascular pseudo parkinsonism

○ Normal pressure hydrocephalus

Key Learning Points

Neurology

- PSP presents with symmetrical stiffness and bradykinesia associated with poor postural reflexes and impaired down-gaze.

Explanation

This patient presents with symmetrical stiffness and bradykinesia associated with poor postural reflexes and impaired down-gaze.

These features are consistent with progressive supra-nuclear palsy (PSP).

MRI imaging may reveal evidence of midbrain atrophy.

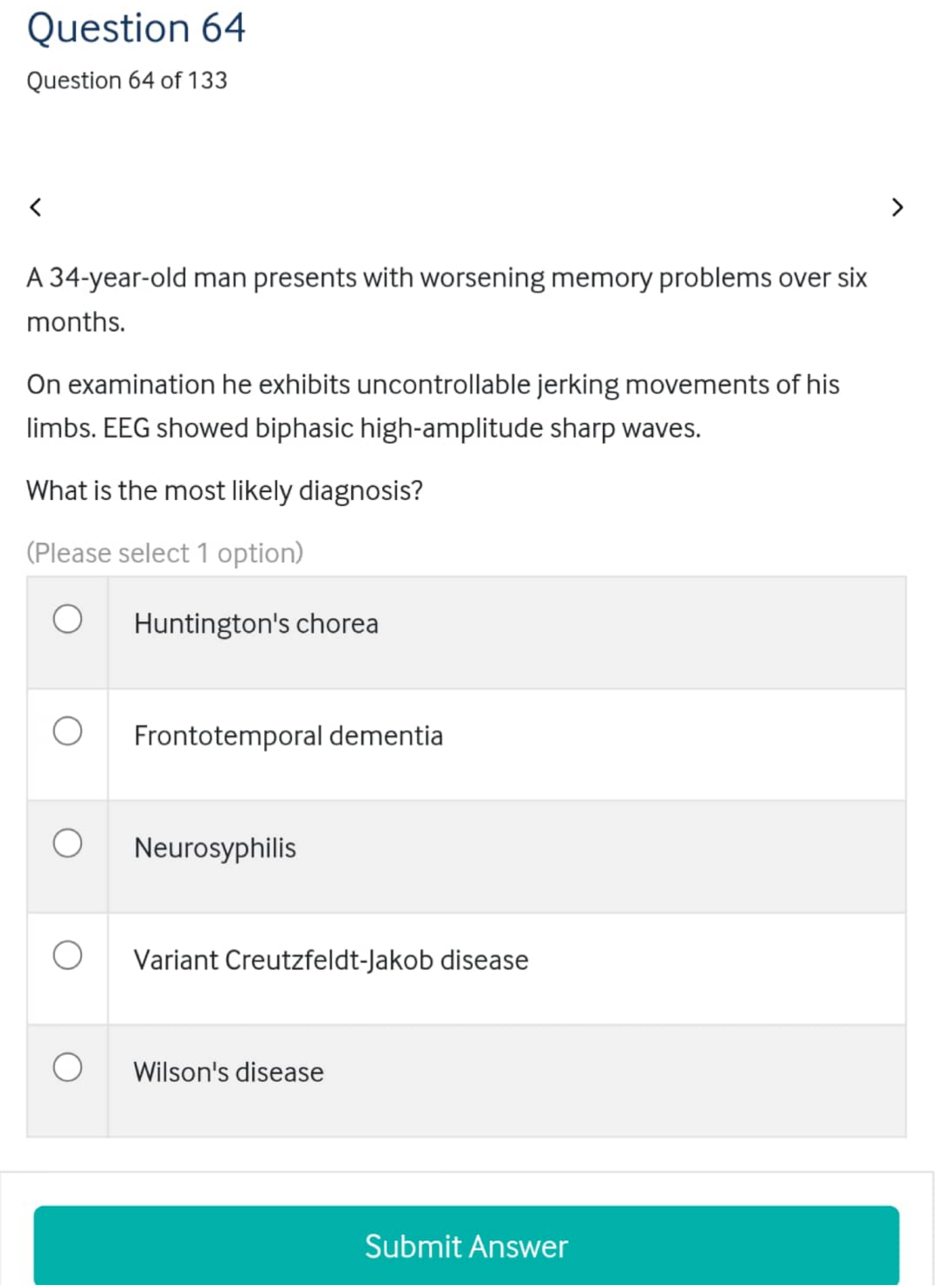
There is a variable response to levodopa.

Brainstem encephalitis would present with an altered conscious level and cranial nerve abnormalities such as oscillopsia, diplopia, facial numbness, dysarthria, hearing loss, and dysphagia.

Idiopathic Parkinson's disease presents with tremor and asymmetrical hemi-parkinsonism.

Vascular parkinsonism presents as lower limb parkinsonism.

Normal pressure hydrocephalus presents with ataxic gait, dementia and urinary incontinence.



Question 64

Question 64 of 133

A 34-year-old man presents with worsening memory problems over six months.

On examination he exhibits uncontrollable jerking movements of his limbs. EEG showed biphasic high-amplitude sharp waves.

What is the most likely diagnosis?

(Please select 1 option)

○ Huntington's chorea

○ Frontotemporal dementia

○ Neurosyphilis

○ Variant Creutzfeldt-Jakob disease

○ Wilson's disease

○ Neurosyphilis

● Variant Creutzfeldt-Jakob disease Correct

○ Wilson's disease

Key Learning Points

Neurology

- The young age of the patient, rapid onset of dementia and presence of myoclonic jerks and EEG are characteristic of Creutzfeldt-Jakob disease.

Explanation

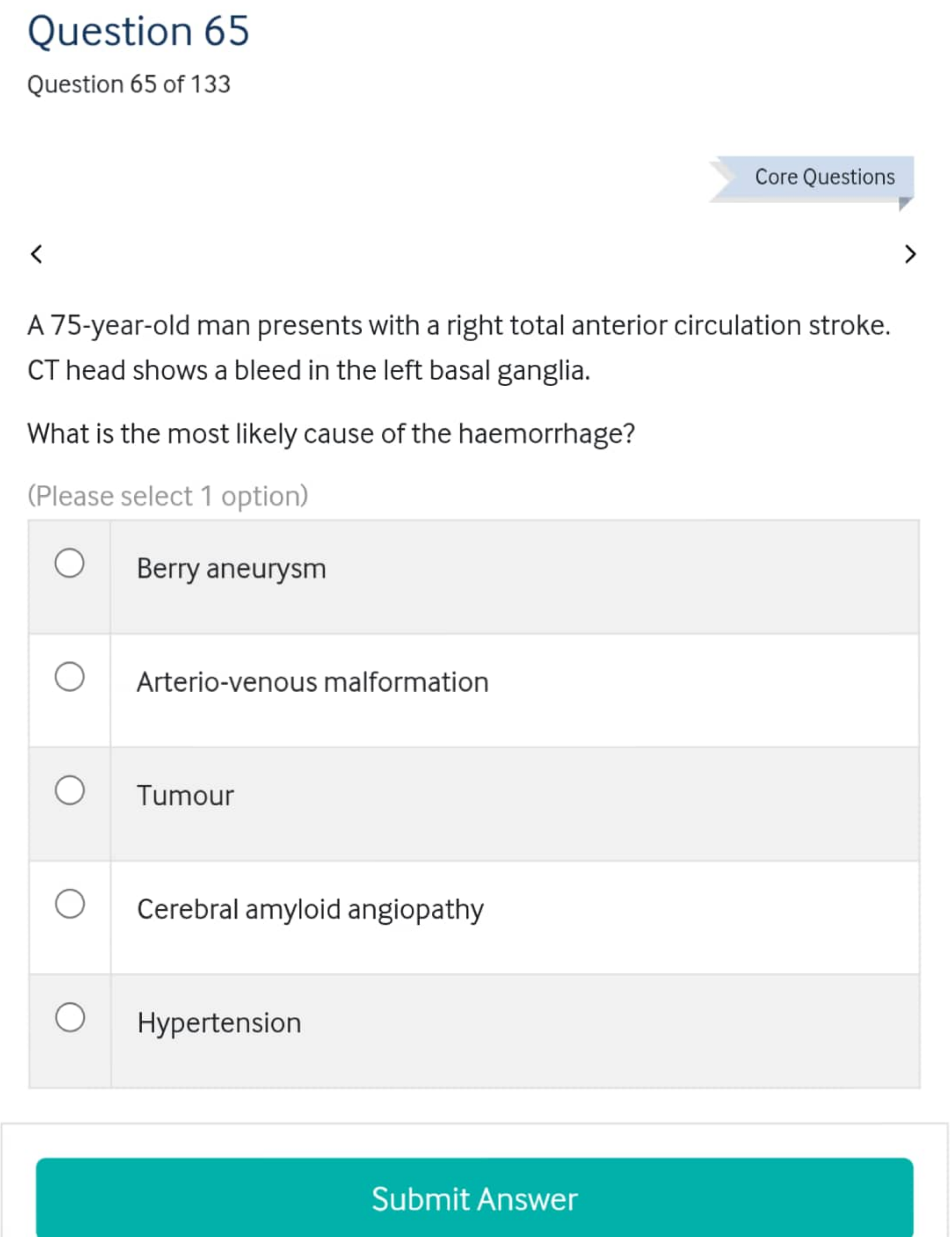
Frontotemporal dementia presents with changes in behaviour and loss of insight more commonly than memory problems.

Huntington's chorea is a neurodegenerative disorder that begins with incoordination and an unsteady gait. As the disease progresses affected individuals develop uncoordinated jerky body movements, cognitive impairment and psychiatric problems. The EEG findings described are not present in this condition.

Neurosyphilis is an infection of the brain and spinal cord in people with untreated syphilis and presents with abnormal gait, incoordination, dementia, seizures, tremors, muscle weakness and incontinence.

Wilson's disease is associated with extrapyramidal features (tremor, parkinsonism, dystonia), dysarthria and psychiatric features.

Next Question



Question 65

Question 65 of 133

A 75-year-old man presents with a right total anterior circulation stroke. CT head shows a bleed in the left basal ganglia.

What is the most likely cause of the haemorrhage?

(Please select 1 option)

○ Berry aneurysm

○ Arterio-venous malformation

○ Tumour

○ Cerebral amyloid angiopathy

○ Hypertension

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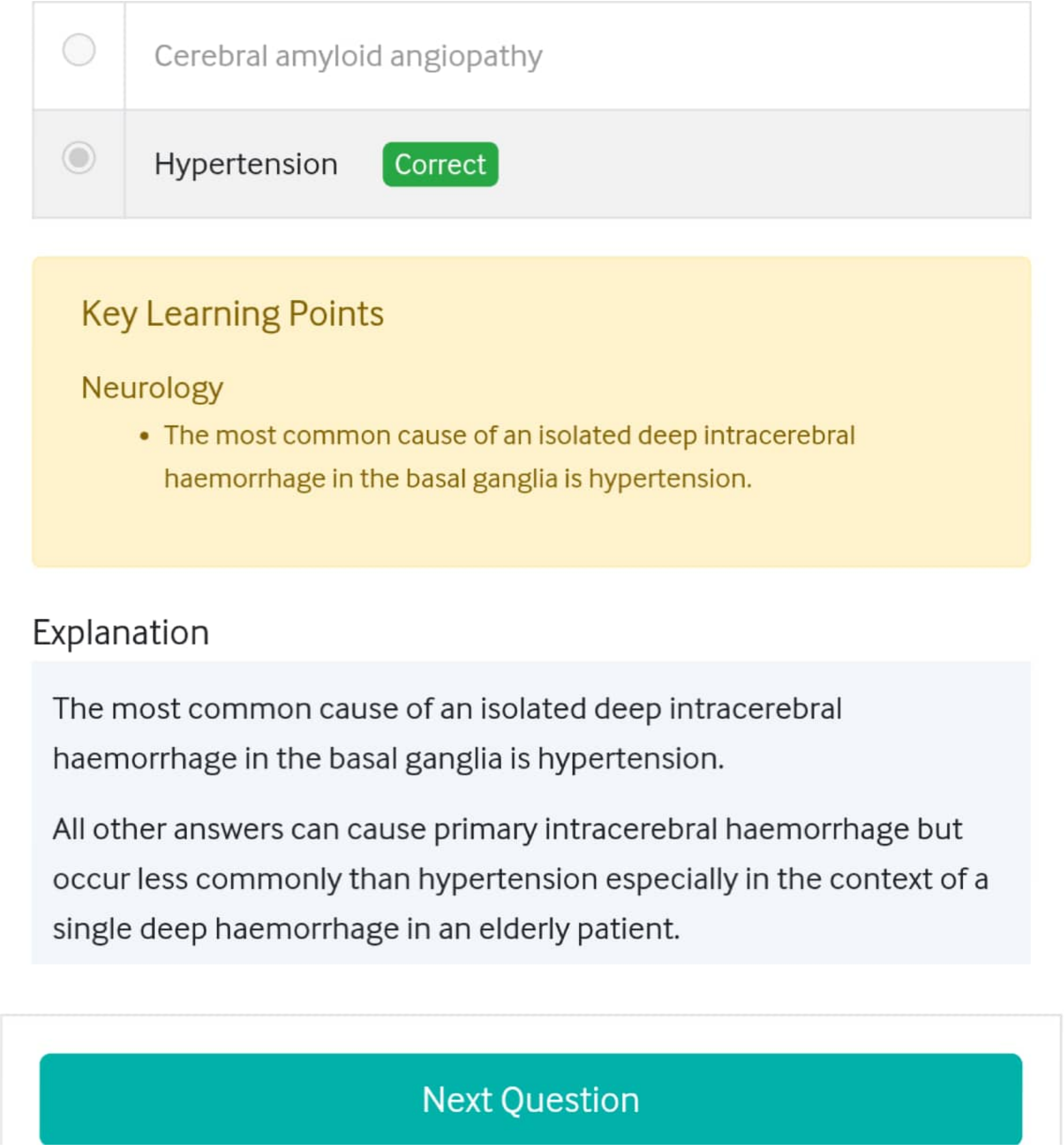


○ Cerebral amyloid angiopathy

● Hypertension

Key Learning Points

Neurology

- The most common cause of an isolated deep intracerebral haemorrhage in the basal ganglia is hypertension.

Explanation

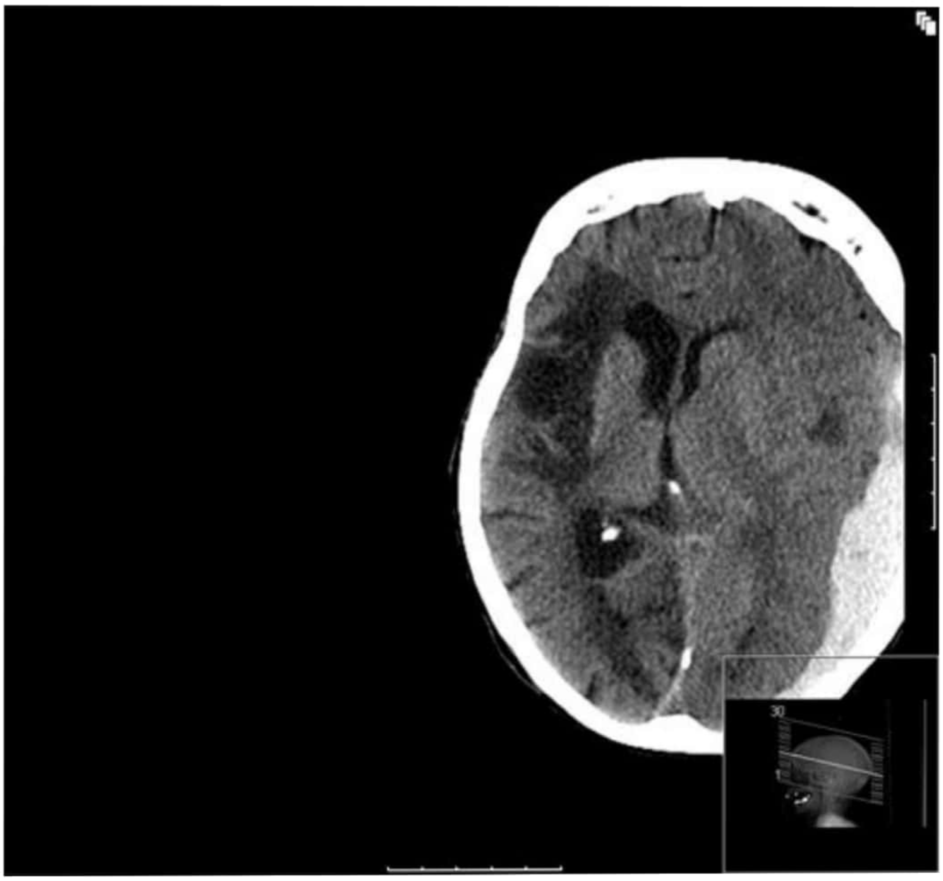
The most common cause of an isolated deep intracerebral haemorrhage in the basal ganglia is hypertension.

All other answers can cause primary intracerebral haemorrhage but occur less commonly than hypertension especially in the context of a single deep haemorrhage in an elderly patient.

A 79-year-old man presents with a sudden onset right-sided weakness and drowsiness. He has a previous history of atrial fibrillation and is taking warfarin.

On examination the GCS is 13/15 and there is a spastic paraparesis of the right hand side with an upgoing right-sided plantar response. An urgent CT scan of the head is performed.

What is the most likely underlying diagnosis?



(Please select 1 option)

○ Acute middle cerebral artery infarct

○ Cerebral abscess

○ Extradural haematoma

○ Intracranial haematoma

○ Subdural haemorrhage

Submit Answer

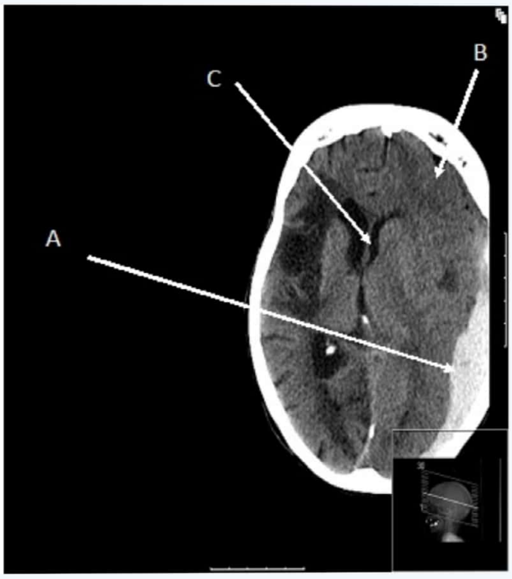
○ Subdural haemorrhage **Correct**

Key Learning Points

Neurology

- Acute subdural haemorrhage is characterised by a convex peripheral high attenuation area with effacement of the sulci and mass effect.

Explanation



The CT demonstrates a large, peripheral high attenuation area in the left cerebral hemisphere extending from the temporoparietal region around to the occipital area (A). The medial border has a predominantly convex appearance. The appearances are consistent with an acute subdural haemorrhage. There is evidence of significant mass effect with effacement of the left hemispheric sulci (B), compression of the lateral ventricles and deviation of structures across the midline away from the haematoma (C).

Acute cerebral infarcts are typically hypodense with vasogenic oedema, they conform to arterial blood supply territories and are typically wedge-shaped (when not lacunar).

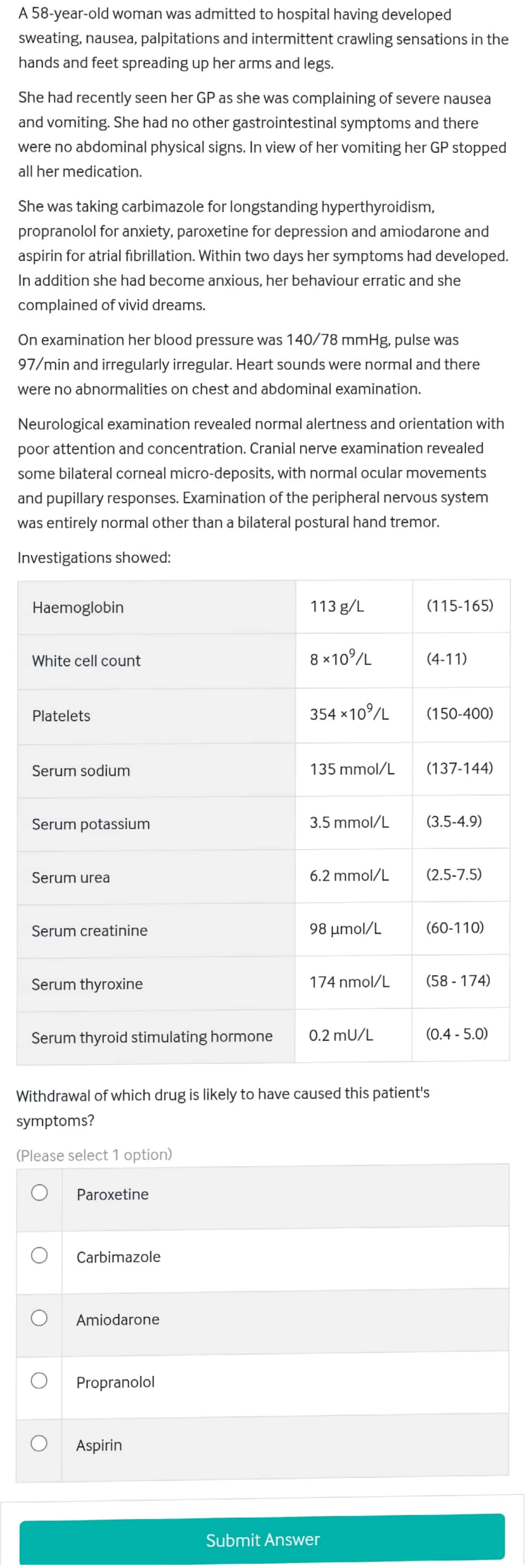
Cerebral abscesses are usually hypodense with surrounding oedema, peripheral ring enhancement is characteristically seen when contrast is administered.

Acute extradural and subdural haematomas would both be high attenuation and anatomically located next to the skull - extradural haematomas have a convex border whilst subdural haematomas have a concave border. In this case the border is predominantly concave and the patient's age and lack of a history of trauma (usually associated with extradural haemorrhage) make a subdural haemorrhage more likely.

Acutely blood is high attenuation (bright) on CT, as time passes the degree of attenuation decreases and the area eventually becomes hypodense. Care must be taken not to miss bleeds when they are isodense with cerebral tissue.

In intracerebral haemorrhage blood is visible within the brain parenchyma.

Next Question



A 58-year-old woman was admitted to hospital having developed sweating, nausea, palpitations and intermittent crawling sensations in the hands and feet spreading up her arms and legs.

She had recently seen her GP as she was complaining of severe nausea and vomiting. She had no other gastrointestinal symptoms and there were no abdominal physical signs. In view of her vomiting her GP stopped all her medication.

She was taking carbimazole for longstanding hyperthyroidism, propranolol for anxiety, paroxetine for depression and amiodarone and aspirin for atrial fibrillation. Within two days her symptoms had developed. In addition she had become anxious, her behaviour erratic and she complained of vivid dreams.

On examination her blood pressure was 140/78 mmHg, pulse was 97/min and irregularly irregular. Heart sounds were normal and there were no abnormalities on chest and abdominal examination.

Neurological examination revealed normal alertness and orientation with poor attention and concentration. Cranial nerve examination revealed some bilateral corneal micro-deposits, with normal ocular movements and pupillary responses. Examination of the peripheral nervous system was entirely normal other than a bilateral postural hand tremor.

Investigations showed:

Haemoglobin	113 g/L	(115-165)
White cell count	8 ×10^9/L	(4-11)
Platelets	354 ×10^9/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.5 mmol/L	(3.5-4.9)
Serum urea	6.2 mmol/L	(2.5-7.5)
Serum creatinine	98 µmol/L	(60-110)
Serum thyroxine	174 nmol/L	(58 - 174)
Serum thyroid stimulating hormone	0.2 mU/L	(0.4 - 5.0)

Withdrawal of which drug is likely to have caused this patient's symptoms?

(Please select 1 option)

- ○ Paroxetine
- ○ Carbimazole
- ○ Amiodarone
- ○ Propranolol
- ○ Aspirin

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(Please select 1 option)

● Paroxetine **Correct**

○ Carbimazole

○ Amiodarone

○ Propranolol

○ Aspirin

Key Learning Points

Neurology, Pharmacology

- Selective serotonin reuptake inhibitor (SSRI) discontinuation syndrome occurs following abrupt withdrawal of SSRIs and symptoms occur after days

Explanation

This patient has presented with selective serotonin reuptake inhibitor (SSRI) discontinuation syndrome which typically occurs following abrupt withdrawal of SSRIs.

Symptoms can occur within days and can last months following withdrawal and then disappear.

A combination of the following symptoms can develop:

- Psychiatric (anxiety, insomnia, mood lability, vivid dreams)
- Gastrointestinal (nausea, vomiting), and
- Neurological (dizziness, headache, paraesthesia, dystonia, tremor).

It is thought that shorter acting SSRIs, when discontinued or when the dosage is lowered, produce an "anticholinergic rebound," interrupting the production of the acetylcholine.

No specific treatment is given.

This patient is on a cocktail of medications for hyperthyroidism. Amiodarone although in this context used for atrial fibrillation can affect thyroid function causing both hyper- and hypothyroidism. Although some of the features would fit with hyperthyroidism, her blood levels of thyroxine are within normal limits.

Paraesthesia as a symptom of thyroid disease is more likely in hypothyroidism complicated by peripheral neuropathy.

Next Question

Question 68

Question 68 of 133

‹ ›

A 40-year-old man with a long history of alcohol abuse is admitted with an acute illness, comprising headache, fever, meningism and ataxia.

MRI brain showed patchy high signal abnormality of the brain stem. CSF analysis showed polymorphonuclear pleocytosis and low glucose. He had failed to improve after three days of intravenous cefotaxime treatment.

What is the most likely causative organism?

(Please select 1 option)

○ *Cryptococcus neoformans*

○ *Nocardia asteroides*

○ *Mycobacterium tuberculosis*

○ *Listeria monocytogenes*

○ *Staphyloccus aureus*

Submit Answer

● *Listeria monocytogenes* Correct

○ *Staphyloccus aureus*

Key Learning Points

Infectious Diseases, Neurology

- Listeria meningitis should always be considered in patients with meningitis associated with brain stem involvement, and in immunosuppressed patients. The treatment of choice is gentamicin and ampicillin.

Explanation

Listeria meningitis should always be considered in patients with meningitis associated with brain stem involvement, and in immunosuppressed patients. The treatment of choice is ampicillin with gentamicin added for synergy. Gentamicin may be stopped after a week if clinical improvement.

Neutrophils usually predominate in the cerebrospinal fluid (CSF) in patients with bacterial meningitis. However, a lymphocytosis is seen in approximately 10% of patients. Lymphocytes may predominate in 30% of patients with meningitis caused by Gram negative bacilli, or in *Listeria monocytogenes* infection.

Note that this means that 70% of patients with *Listeria* meningitis will have a neutrophilic CSF.

A lymphocytic CSF predominates in TB and fungal meningitis.

Next Question

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A 70-year-old man presented to the Emergency department with weakness affecting both legs.

The weakness had been preceded by a sudden onset of left sided hip pain, which had become intractable over the last four hours. He had also developed numbness over both legs to the level of the sternum. He had a past medical history of hypertension and ischaemic heart disease and took atenolol and Suscard buccal when required. He was a smoker of 10 cigarettes per day and drank a whiskey a night.

On examination he appeared in distress. His blood pressure was 167/89 mmHg, pulse was 88/min and regular and temperature was 35.8°C. Auscultation of the heart was normal.

On examination of the cranial nerves there were no abnormalities to be found. Fundoscopy revealed some bilateral silver wiring and AV-nipping. Upper limb examination was normal. On examination of the lower limb, tone was decreased bilaterally, with pyramidal weakness of 2/5. Sensory examination revealed reduced pinprick and temperature sensation to the level of T6. He was also noted to be in urinary retention.

Investigations revealed:

Haemoglobin	139 g/L	(130-180)
White cell count	11.0 $\times 10^9$/L	(4-11)
Platelets	345 $\times 10^9$/L	(150-400)
Serum sodium	142 mmol/L	(137-144)
Serum potassium	3.9 mmol/L	(3.5-4.9)
Serum urea	7.8 mmol/L	(2.5-7.5)
Serum creatinine	134 μmol/L	(60-110)
Fasting plasma glucose	8.9 mmol/L	(3-6)
Serum cholesterol	5.9 mmol/L	(< 5.2)

An x ray of the spine showed osteoarthritis affecting vertebrae C7-T1 and L4-5.

What is the likely cause for this patient's symptoms?

(Please select 1 option)

○	Anterior cord syndrome secondary to cervical disc herniation at C6/7
○	Anterior spinal artery occlusion
○	Central cord syndrome secondary to osteoarthritis
○	Transverse myelitis
○	Parasagittal meningioma

CamScanner الممسوحة ضوئيا بـ

● Anterior spinal artery occlusion **Correct**

○ Central cord syndrome secondary to osteoarthritis

○ Transverse myelitis

○ Parasagittal meningioma

Key Learning Points

Neurology

- Sudden onset of anterior cord syndrome causing bilateral paresis is suggestive of an anterior spinal artery lesion.

Explanation

This patient presents with sudden onset of anterior cord syndrome, affecting the legs. There is spastic weakness in association with spinothalamic disruption.

He has several risk factors for arteriosclerosis including:

- hypertension
- hypercholesterolaemia
- diabetes, and
- ischaemic heart disease.

The likely cause is anterior spinal artery infarct at the T4-T6 watershed, which would fit with the sensory level found on examination.

Disc herniation or a central cord syndrome in the cervical region would cause some upper limb signs, depending on the level, which is not the case in this gentleman.

A parasagittal meningioma is unlikely given the acute onset associated with pain, in absence of headache.

A transverse myelitis would be unusual in a patient of this age group.

Next Question

A 62-year-old man presents with paraesthesia and numbness in both lateral aspects of the hands over the past one year.

He has also been aware of some weakness when using his thumbs. He has also has a reduced appetite and feels full on small meals. Consequently he has lost some weight over the last six months.

Similarly, he describes episodes of diarrhoea which can occur at night and have concerned him. He has also been aware of increasing urinary frequency. He has a past history of hypertension for which he takes bendroflumethiazide and ramipril. He is a smoker of 20 cigarettes per day and drinks approximately 10 units of alcohol, weekly. He is employed as a carpenter and has a wife and grown up children.

On examination, his pulse is 72 bpm, he has blood pressure is 134/86 mmHg and this is associated with a drop on standing by 40 mmHg. His BMI is 24.5 kg/m^2. Examination of his hands reveals sensory loss over the lateral three and a half fingers bilaterally with some weakness of the thenar muscles. There is also loss of sensation in both feet and there is loss of ankle jerks and plantar responses are equivocal.

Select the most likely diagnosis.

(Please select 1 option)

○	Diabetes mellitus
○	Cervical spondylolytic myelopathy
○	Alcohol neuropathy
○	Kearns-Sayre-Daroff syndrome
○	Dorsal cord meningiomas

Submit Answer

○ Diabetes mellitus **This is the correct answer**

● Cervical spondylolytic myelopathy **Incorrect answer selected**

○ Alcohol neuropathy

○ Kearns-Sayre-Daroff syndrome

○ Dorsal cord meningiomas

Key Learning Points

Neurology

- Differential diagnosis for autonomic neuropathy includes DM, CRF, AIDS, AL, Porphyria

Explanation

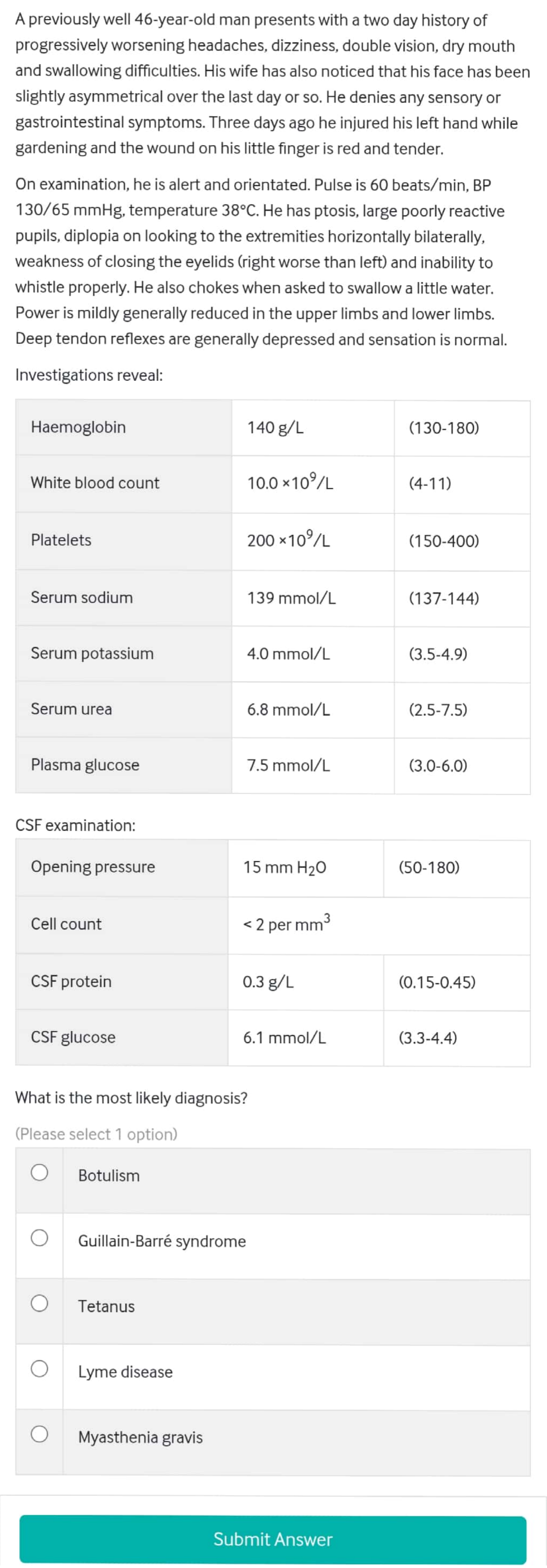
This man has:

- Autonomic neuropathy
- Compression neuropathy (bilateral carpal tunnel syndrome), and
- Peripheral sensory neuropathy (bilateral foot pain).

The differential diagnosis of autonomic neuropathy is small:

- Diabetes
- Chronic renal failure
- AIDS
- Primary amyloidosis, and
- Porphyria.

It seems from his symptoms that this could be diabetes.



A previously well 46-year-old man presents with a two day history of progressively worsening headaches, dizziness, double vision, dry mouth and swallowing difficulties. His wife has also noticed that his face has been slightly asymmetrical over the last day or so. He denies any sensory or gastrointestinal symptoms. Three days ago he injured his left hand while gardening and the wound on his little finger is red and tender.

On examination, he is alert and orientated. Pulse is 60 beats/min, BP 130/65 mmHg, temperature 38°C. He has ptosis, large poorly reactive pupils, diplopia on looking to the extremities horizontally bilaterally, weakness of closing the eyelids (right worse than left) and inability to whistle properly. He also chokes when asked to swallow a little water. Power is mildly generally reduced in the upper limbs and lower limbs. Deep tendon reflexes are generally depressed and sensation is normal.

Investigations reveal:

Haemoglobin	140 g/L	(130-180)
White blood count	10.0 $\times 10^9$/L	(4-11)
Platelets	200 $\times 10^9$/L	(150-400)
Serum sodium	139 mmol/L	(137-144)
Serum potassium	4.0 mmol/L	(3.5-4.9)
Serum urea	6.8 mmol/L	(2.5-7.5)
Plasma glucose	7.5 mmol/L	(3.0-6.0)

CSF examination:

Opening pressure	15 mm H_2O	(50-180)
Cell count	< 2 per mm^3	
CSF protein	0.3 g/L	(0.15-0.45)
CSF glucose	6.1 mmol/L	(3.3-4.4)

What is the most likely diagnosis?

(Please select 1 option)

○ Botulism

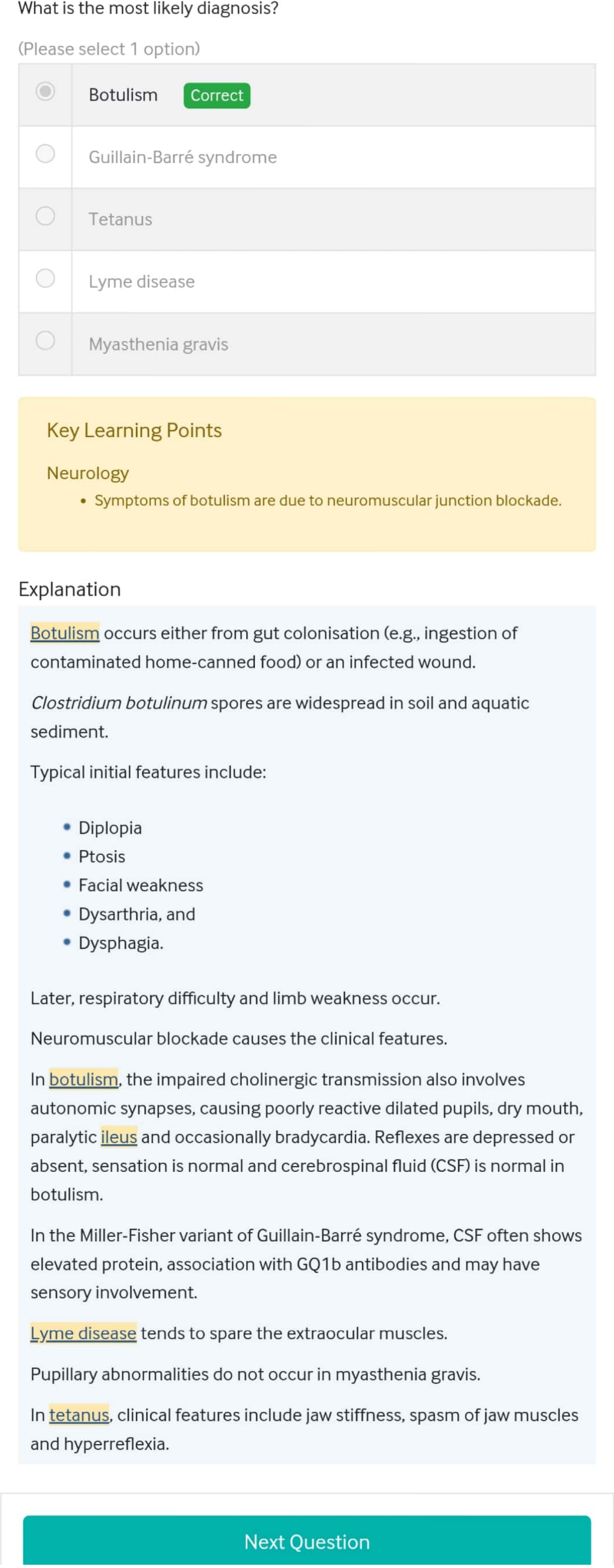
○ Guillain-Barré syndrome

○ Tetanus

○ Lyme disease

○ Myasthenia gravis

Submit Answer



What is the most likely diagnosis?

(Please select 1 option)

● Botulism **Correct**

○ Guillain-Barré syndrome

○ Tetanus

○ Lyme disease

○ Myasthenia gravis

Key Learning Points

Neurology

- Symptoms of botulism are due to neuromuscular junction blockade.

Explanation

Botulism occurs either from gut colonisation (e.g., ingestion of contaminated home-canned food) or an infected wound.

Clostridium botulinum spores are widespread in soil and aquatic sediment.

Typical initial features include:

- Diplopia
- Ptosis
- Facial weakness
- Dysarthria, and
- Dysphagia.

Later, respiratory difficulty and limb weakness occur.

Neuromuscular blockade causes the clinical features.

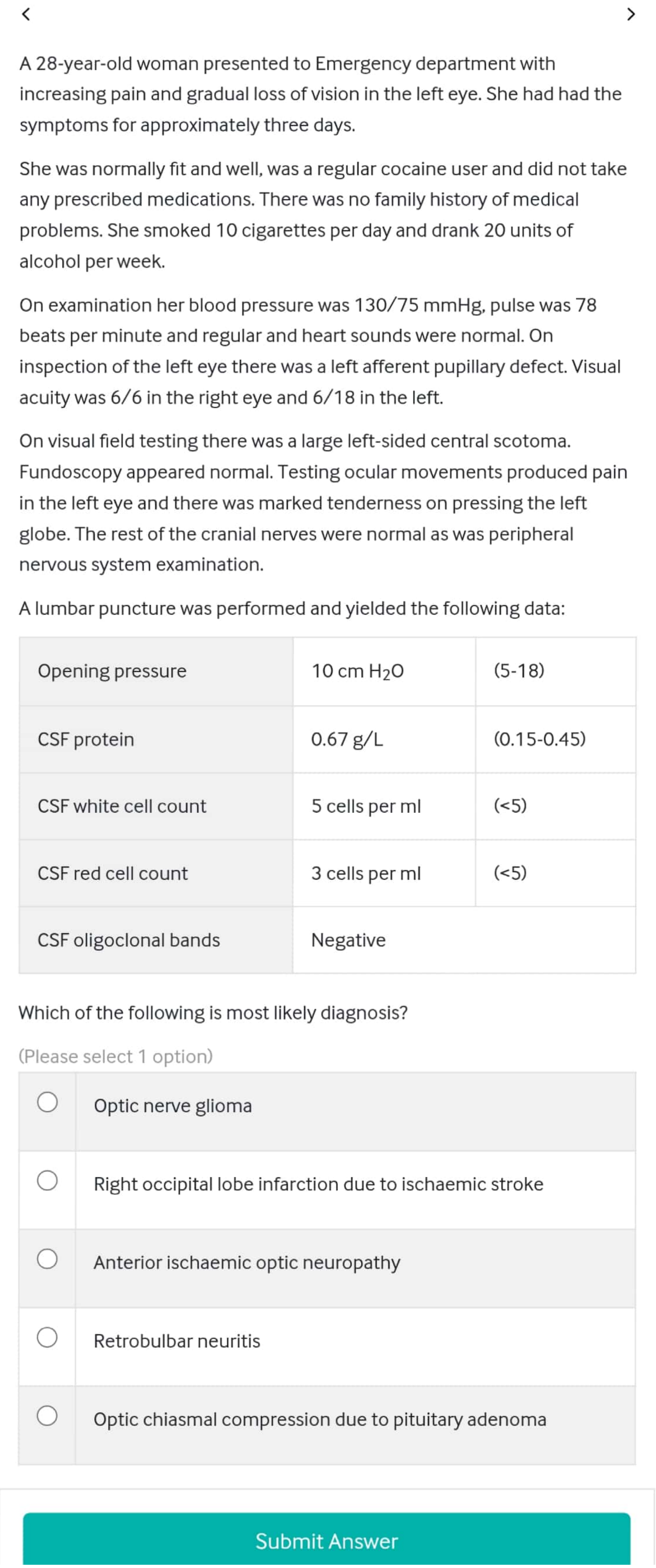
In botulism, the impaired cholinergic transmission also involves autonomic synapses, causing poorly reactive dilated pupils, dry mouth, paralytic ileus and occasionally bradycardia. Reflexes are depressed or absent, sensation is normal and cerebrospinal fluid (CSF) is normal in botulism.

In the Miller-Fisher variant of Guillain-Barré syndrome, CSF often shows elevated protein, association with GQ1b antibodies and may have sensory involvement.

Lyme disease tends to spare the extraocular muscles.

Pupillary abnormalities do not occur in myasthenia gravis.

In tetanus, clinical features include jaw stiffness, spasm of jaw muscles and hyperreflexia.



A 28-year-old woman presented to Emergency department with increasing pain and gradual loss of vision in the left eye. She had had the symptoms for approximately three days.

She was normally fit and well, was a regular cocaine user and did not take any prescribed medications. There was no family history of medical problems. She smoked 10 cigarettes per day and drank 20 units of alcohol per week.

On examination her blood pressure was 130/75 mmHg, pulse was 78 beats per minute and regular and heart sounds were normal. On inspection of the left eye there was a left afferent pupillary defect. Visual acuity was 6/6 in the right eye and 6/18 in the left.

On visual field testing there was a large left-sided central scotoma. Fundoscopy appeared normal. Testing ocular movements produced pain in the left eye and there was marked tenderness on pressing the left globe. The rest of the cranial nerves were normal as was peripheral nervous system examination.

A lumbar puncture was performed and yielded the following data:

Opening pressure	10 cm H_2O	(5-18)
CSF protein	0.67 g/L	(0.15-0.45)
CSF white cell count	5 cells per ml	(<5)
CSF red cell count	3 cells per ml	(<5)
CSF oligoclonal bands	Negative	

Which of the following is most likely diagnosis?

(Please select 1 option)

○ Optic nerve glioma

○ Right occipital lobe infarction due to ischaemic stroke

○ Anterior ischaemic optic neuropathy

○ Retrobulbar neuritis

○ Optic chiasmal compression due to pituitary adenoma

● Retrobulbar neuritis **Correct**

○ Optic chiasmal compression due to pituitary adenoma

Key Learning Points

Neurology

- Cases of mono-neuritits in a young female patient should prompt investigation for multiple sclerosis.

Explanation

The patient's age, length of history and pain in the eye all favour optic neuritis.

Pituitary or chiasmal lesions usually lead to a bitemporal hemianopia.

Whilst optic nerve gliomas can present with visual failure and an afferent papillary defect, the visual loss would be expected to occur over a much longer period.

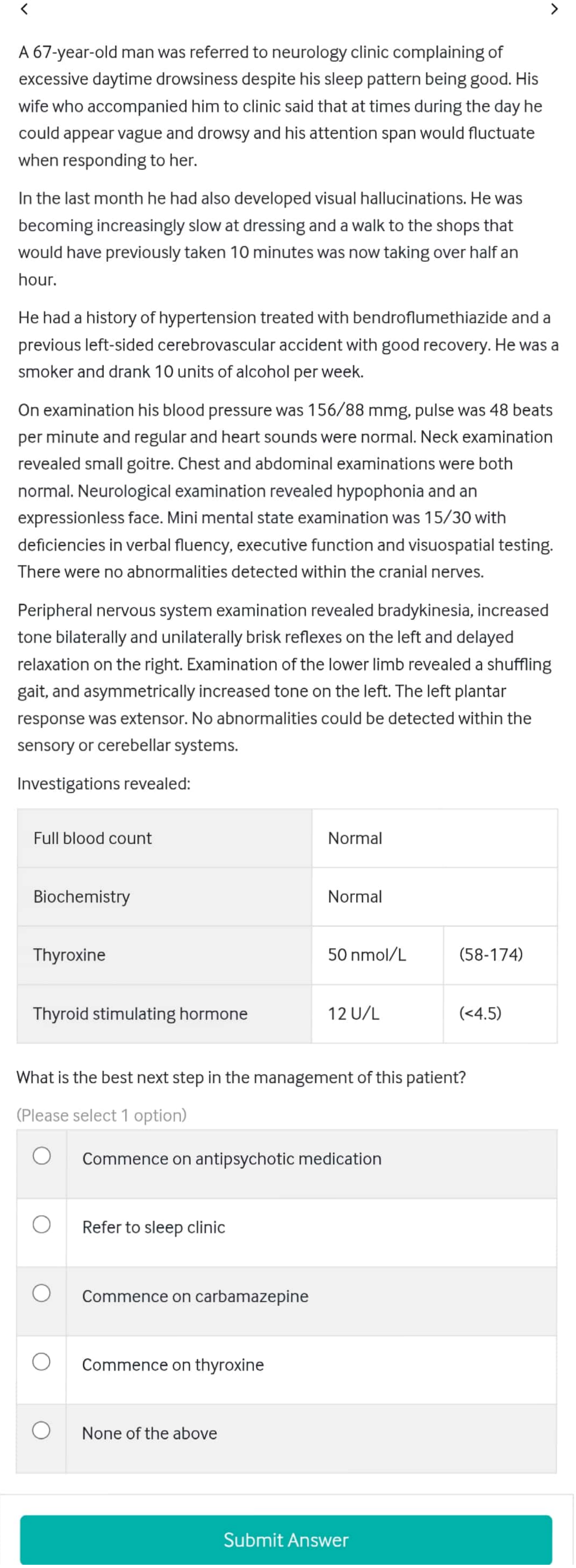
Occipital lobe infarction is unlikely at this age although it should be considered in cocaine users, but the visual defect would be a macular sparing left homonymous hemianopia.

Anterior ischaemic optic neuropathy causes persistent monocular visual loss associated with a relative afferent pupillary defect and is the most common cause in patients over the age of 50 years. There is normally a background of hypertension and diabetes.

Further Reading:

Friends with MS. Retrobulbar Neuritis.

Next Question



A 67-year-old man was referred to neurology clinic complaining of excessive daytime drowsiness despite his sleep pattern being good. His wife who accompanied him to clinic said that at times during the day he could appear vague and drowsy and his attention span would fluctuate when responding to her.

In the last month he had also developed visual hallucinations. He was becoming increasingly slow at dressing and a walk to the shops that would have previously taken 10 minutes was now taking over half an hour.

He had a history of hypertension treated with bendroflumethiazide and a previous left-sided cerebrovascular accident with good recovery. He was a smoker and drank 10 units of alcohol per week.

On examination his blood pressure was 156/88 mmg, pulse was 48 beats per minute and regular and heart sounds were normal. Neck examination revealed small goitre. Chest and abdominal examinations were both normal. Neurological examination revealed hypophonia and an expressionless face. Mini mental state examination was 15/30 with deficiencies in verbal fluency, executive function and visuospatial testing. There were no abnormalities detected within the cranial nerves.

Peripheral nervous system examination revealed bradykinesia, increased tone bilaterally and unilaterally brisk reflexes on the left and delayed relaxation on the right. Examination of the lower limb revealed a shuffling gait, and asymmetrically increased tone on the left. The left plantar response was extensor. No abnormalities could be detected within the sensory or cerebellar systems.

Investigations revealed:

Full blood count	Normal	
Biochemistry	Normal	
Thyroxine	50 nmol/L	(58-174)
Thyroid stimulating hormone	12 U/L	(<4.5)

What is the best next step in the management of this patient?

(Please select 1 option)

○ Commence on antipsychotic medication

○ Refer to sleep clinic

○ Commence on carbamazepine

○ Commence on thyroxine

○ None of the above

● Commence on thyroxine **Correct**

○ None of the above

Key Learning Points

Neurology

- Hypothyroidism can sometimes mimic parkinsonian disorders.

Explanation

The most likely cause for this patient's symptoms is diffuse Lewy body disease (DLB).

He has parkinsonian features associated with fluctuating cognitive functioning and visual hallucinations. The cognitive deficits manifest as problems with executive functioning, verbal fluency and visuospatial tasks.

Other symptoms which can occur in DLB (versus AD) include non-visual hallucinations, delusions and unexplained syncope. Further tests including imaging, neuropsychological testing will aid in the assessment of cognitive decline and the type of dementia.

However, the presence of low thyroxine level and elevated thyroid-stimulating hormone (>10) are consistent with a diagnosis of hypothyroidism.

He also has slow pulse rate, small goitre and delayed relaxation of right-sided reflexes (pseudo-myotonia). These findings together with his daytime drowsiness warrant a trial of thyroxine. In view of his age and history of vascular disease, a small dose of oral thyroxine should be started and review after several months to see if any improvement occurs in his clinical state.

The history does not suggest partial seizures as the patient appears responsive during the episodes of cognitive fluctuation. Day-time somnolence is a problem with sleep apnoea; however the intermittent nature of symptoms and other neurological findings would not be explained by sleep apnoea.

None of his features warrant anti-psychotic medication, which has recently been shown to be over-prescribed in dementia patients with associated increased morbidity and mortality.

Next Question

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A 22-year-old woman was referred to a neurologist complaining of painful spasms and twisting movements affecting her lower limbs (right more than left).

She had noticed that when walking she could suddenly lurch forwards and lose her balance. These symptoms had come on over a few days. She had also noticed some involuntary head movements and her boyfriend had commented that her speech had become slurred. Preceding her symptoms she had had a sore throat.

Past medical history included a deep vein thrombosis (DVT) at 17 years of age. She had been taking the oral contraceptive pill for two years. There was a family history of stroke and dementia on her mother's side and she was a smoker of 20 cigarettes per day.

On examination she had a marked dysarthria and exhibited a continuous flow of irregular jerky movements affecting both legs (right worse than left). In addition there also appeared to be some abnormal jerky movements of her right arm and head. General and cardiovascular examinations were normal.

MR brain scan was normal.

Blood tests revealed:

Haemoglobin	123 g/L	(115 - 165)
White cell count	8.9 $\times 10^9$/L	(4 - 11)
Platelets	225 $\times 10^9$/L	(150 - 400)
Sodium	134 mmol/L	(137 - 144)
Potassium	3.5 mmol/L	(3.5 - 4.9)
Urea	4.6 mmol/L	(2.5 - 7.5)
Creatinine	78 µmol/L	(60 - 110)
Copper	23 µmol/L	(12 - 26)
Caeruloplasmin	260 mg/L	(200 - 350)
Anti-cardiolipin IgG	20	(0 - 20)
Anti-cardiolipin IgM	18	(0 - 20)
Streptolysin O titre	500	(0 - 200)
Anti-DNAse	1200	(<85)
Anti-basal ganglia antibodies	Positive	-
Thyroxine	60 nmol/L	(58 - 178)
Thyroid stimulating hormone	4.0 mU/L	(0.4 - 5)

Given the history and above findings, how might you best manage this patient?

(Please select 1 option)

○ Penicillin V and sulpiride

○ Refer for psychiatric assessment

○ Refer for genetic testing

○ Anticoagulate with warfarin

○ Discontinue the oral contraceptive pill

Submit Answer

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(Please select 1 option)

● Penicillin V and sulpiride **Correct**

○ Refer for psychiatric assessment

○ Refer for genetic testing

○ Anticoagulate with warfarin

○ Discontinue the oral contraceptive pill

Key Learning Points

Neurology

- Treatment for chorea secondary to strep infection is with tetrabenazine or sulpiride and a course of penicillin for acute infection.

Explanation

This patient presents with acute chorea as a result of streptococcal throat infection or Sydenham's chorea.

This tends to occur in children presenting initially with irritability and inattentiveness. This then progresses to generalised chorea with speech involvement occurring in 20% of cases. Those who have had several attacks in childhood may develop chorea in adulthood when exposed to drugs such as the oral contraceptive pill, phenytoin or digoxin, or during pregnancy.

Treatment is for chorea with tetrabenazine or sulpiride and a course of penicillin for acute infection.

Although there is a history of DVT, antiphospholipid antibody testing was negative.

The oral contraceptive pill should probably be discontinued in view of previous DVT although drug-induced chorea is not the primary diagnosis in this patient.

Huntington's disease tends to present later in life with a progressive history of motor and cognitive signs and symptoms.

There is nothing in the history to suggest an underlying psychiatric illness in this patient.

Next Question

A 50-year-old man with learning difficulties is referred because of urinary incontinence. He lives alone.

Over the last two months, he has been finding it increasingly difficult to manage simple household tasks like making meals and cleaning. He claims his 'concentration is not as good as before.' He had a son who died from complications of multiple sclerosis.

On examination, he is orientated in time, place and person. On memory testing, he was able to register information but was only able to recall not more than three items out of six after five minutes. Long term memory was patchy. On testing serial 3s, he reached 25 after 30 seconds. His thought content was normal, but affect was labile. He was unable to interpret the true meaning of a proverb.

Cranial nerve examination was unremarkable, except for horizontal nystagmus on lateral gaze bilaterally. Examination of the limbs revealed past-pointing in the upper limbs, difficulty with heel to toe walking in the lower limbs, brisk deep tendon reflexes and extensor plantar responses. Palmomental reflexes were present bilaterally.

What treatment would you initiate in this patient?

(Please select 1 option)

○ Paroxetine

○ Rivastigmine

○ Levodopa

○ No treatment

○ Thiamine

Submit Answer

○ Levodopa

○ No treatment

○ Thiamine **This is the correct answer**

Key Learning Points

Neurology

- Korsakoff syndrome is a chronic disorder caused by severe thiamine deficiency. It is characterised by memory problems, confabulation and apathy.

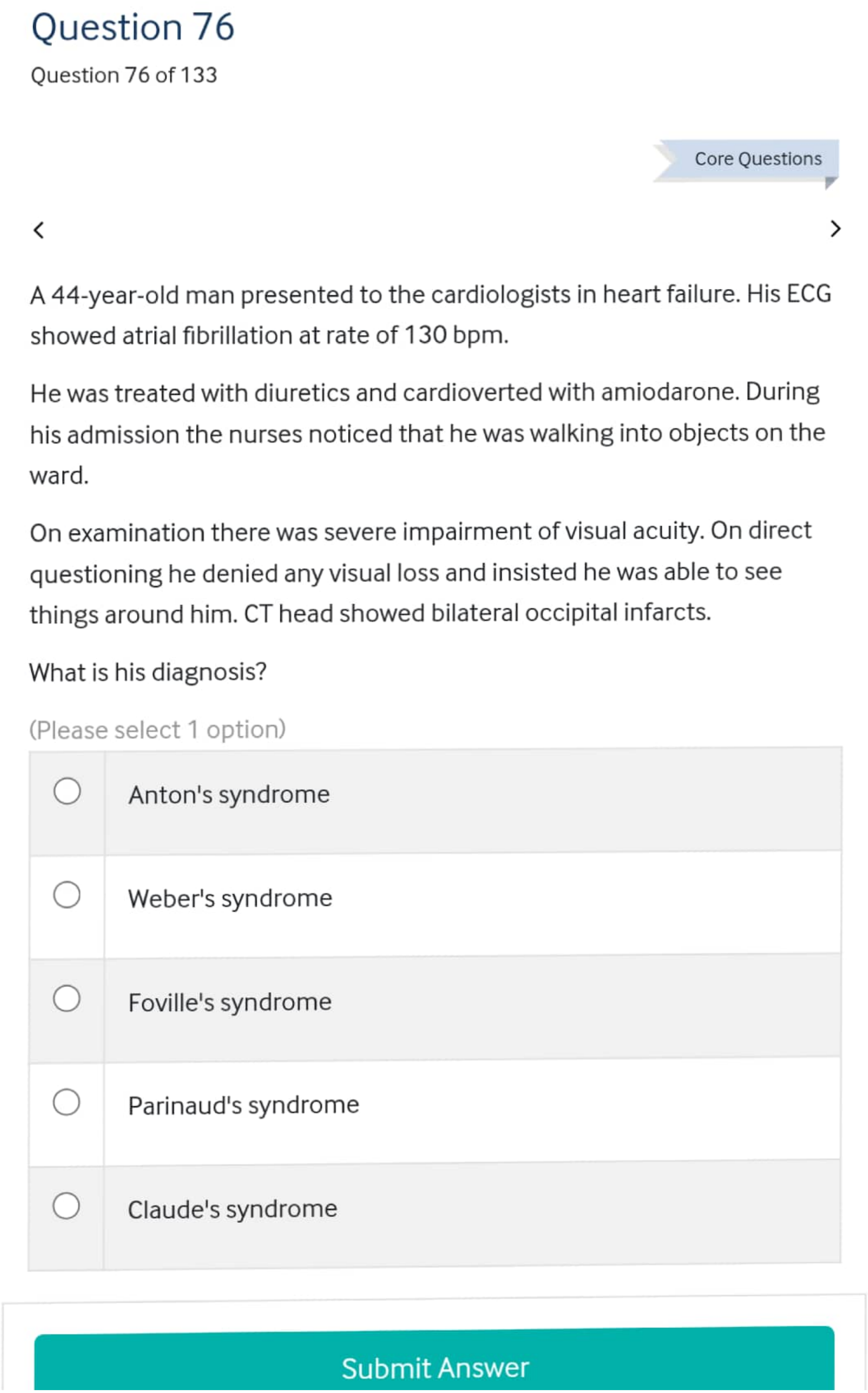
Explanation

The recent onset of memory difficulties, mild impairment of attention and concentration and the lack of ability to reason would be more in keeping with Korsakoff's amnestic syndrome than Alzheimer's disease as the memory impairment would be more severe in the latter.

In addition, the suggestion of retained features of acute Wernicke's encephalopathy, in particular ataxia and nystagmus, add support to the diagnosis of Korsakoff's amnestic syndrome.

Thiamine is given in established cases to prevent progression of deficits.

Next Question



Question 76

Question 76 of 133

A 44-year-old man presented to the cardiologists in heart failure. His ECG showed atrial fibrillation at rate of 130 bpm.

He was treated with diuretics and cardioverted with amiodarone. During his admission the nurses noticed that he was walking into objects on the ward.

On examination there was severe impairment of visual acuity. On direct questioning he denied any visual loss and insisted he was able to see things around him. CT head showed bilateral occipital infarcts.

What is his diagnosis?

(Please select 1 option)

- ○ Anton's syndrome
- ○ Weber's syndrome
- ○ Foville's syndrome
- ○ Parinaud's syndrome
- ○ Claude's syndrome

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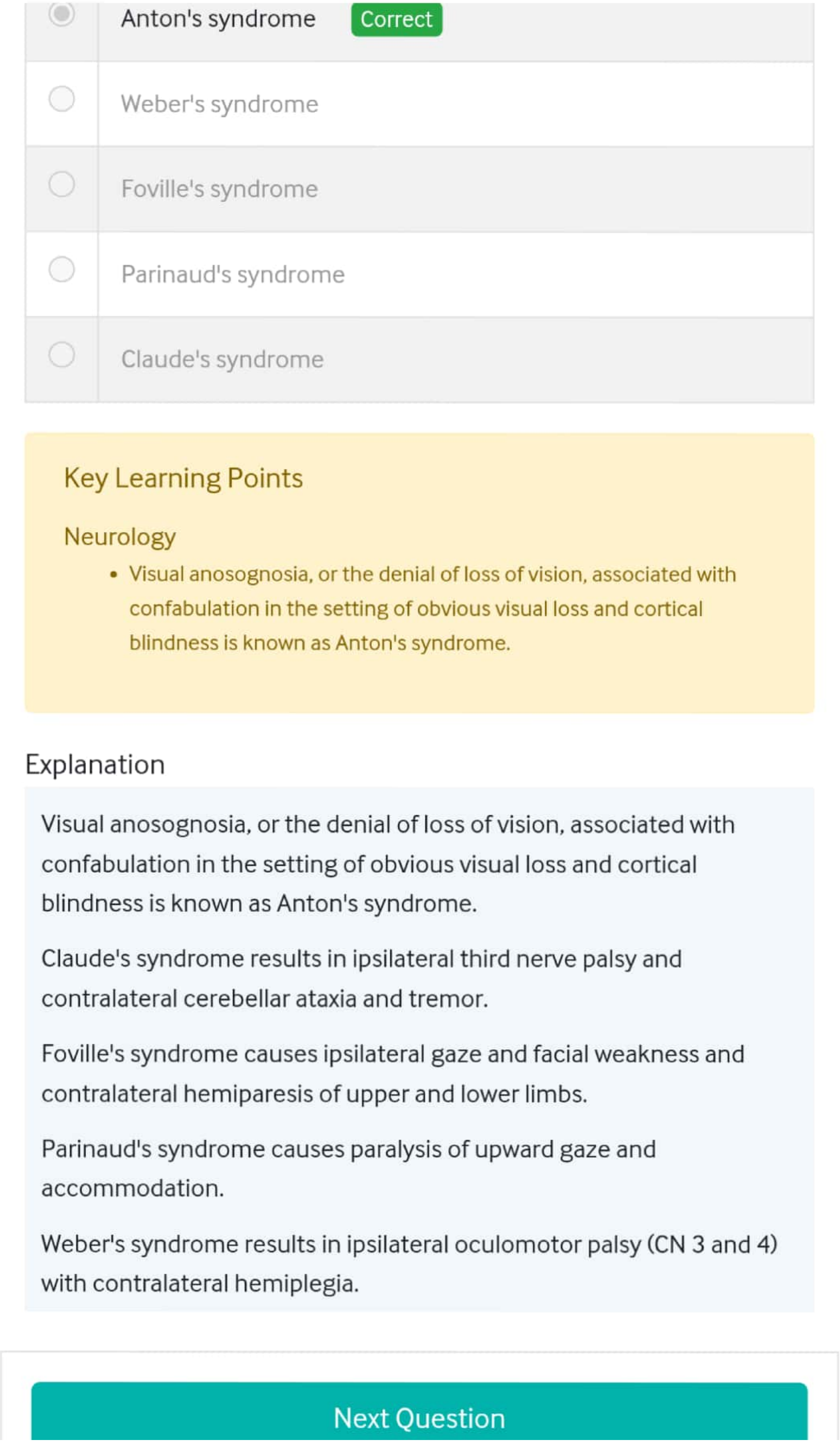

● Anton's syndrome Correct

○ Weber's syndrome

○ Foville's syndrome

○ Parinaud's syndrome

○ Claude's syndrome

Key Learning Points

Neurology

- Visual anosognosia, or the denial of loss of vision, associated with confabulation in the setting of obvious visual loss and cortical blindness is known as Anton's syndrome.

Explanation

Visual anosognosia, or the denial of loss of vision, associated with confabulation in the setting of obvious visual loss and cortical blindness is known as Anton's syndrome.

Claude's syndrome results in ipsilateral third nerve palsy and contralateral cerebellar ataxia and tremor.

Foville's syndrome causes ipsilateral gaze and facial weakness and contralateral hemiparesis of upper and lower limbs.

Parinaud's syndrome causes paralysis of upward gaze and accommodation.

Weber's syndrome results in ipsilateral oculomotor palsy (CN 3 and 4) with contralateral hemiplegia.

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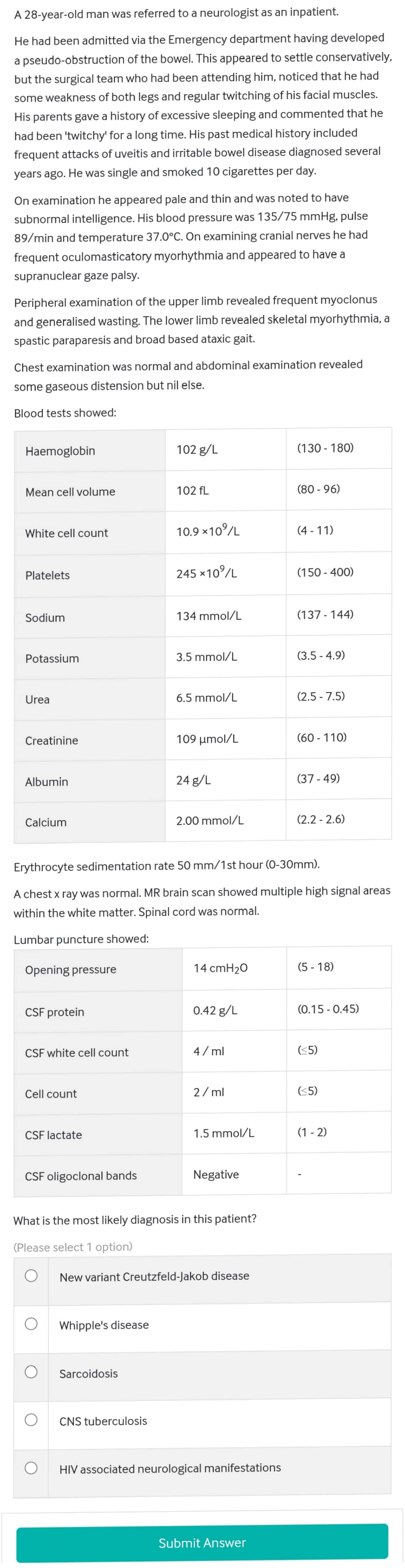


A 28-year-old man was referred to a neurologist as an inpatient.

He had been admitted via the Emergency department having developed a pseudo-obstruction of the bowel. This appeared to settle conservatively, but the surgical team who had been attending him, noticed that he had some weakness of both legs and regular twitching of his facial muscles. His parents gave a history of excessive sleeping and commented that he had been 'twitchy' for a long time. His past medical history included frequent attacks of uveitis and irritable bowel disease diagnosed several years ago. He was single and smoked 10 cigarettes per day.

On examination he appeared pale and thin and was noted to have subnormal intelligence. His blood pressure was 135/75 mmHg, pulse 89/min and temperature 37.0°C. On examining cranial nerves he had frequent oculomasticatory myorhythmia and appeared to have a supranuclear gaze palsy.

Peripheral examination of the upper limb revealed frequent myoclonus and generalised wasting. The lower limb revealed skeletal myorhythmia, a spastic paraparesis and broad based ataxic gait.

Chest examination was normal and abdominal examination revealed some gaseous distension but nil else.

Blood tests showed:

Haemoglobin	102 g/L	(130 - 180)
Mean cell volume	102 fL	(80 - 96)
White cell count	10.9 ×10^9/L	(4 - 11)
Platelets	245 ×10^9/L	(150 - 400)
Sodium	134 mmol/L	(137 - 144)
Potassium	3.5 mmol/L	(3.5 - 4.9)
Urea	6.5 mmol/L	(2.5 - 7.5)
Creatinine	109 µmol/L	(60 - 110)
Albumin	24 g/L	(37 - 49)
Calcium	2.00 mmol/L	(2.2 - 2.6)

Erythrocyte sedimentation rate 50 mm/1st hour (0-30mm).

A chest x ray was normal. MR brain scan showed multiple high signal areas within the white matter. Spinal cord was normal.

Lumbar puncture showed:

Opening pressure	14 cmH_2O	(5 - 18)
CSF protein	0.42 g/L	(0.15 - 0.45)
CSF white cell count	4 / ml	(≤5)
Cell count	2 / ml	(≤5)
CSF lactate	1.5 mmol/L	(1 - 2)
CSF oligoclonal bands	Negative	-

What is the most likely diagnosis in this patient?

(Please select 1 option)

○ New variant Creutzfeld-Jakob disease

○ Whipple's disease

○ Sarcoidosis

○ CNS tuberculosis

○ HIV associated neurological manifestations

○ Whipple's disease **This is the correct answer**

○ Sarcoidosis

○ CNS tuberculosis

○ HIV associated neurological manifestations

Key Learning Points

Neurology

- Duodenal biopsy in Whipples disease shows PAS positive foamy macrophages throughout the lamina propria and the bacillus Tropheryma whippelii

Explanation

This is a difficult case. The clues to a diagnosis of Whipple's disease are:

- A background of malabsorption probably misdiagnosed as irritable bowel disease
- Characteristic oculomasticatory myorhythmia and myoclonus
- Oculoparesis, and
- A suspicion of cognitive and hypothalamic involvement.

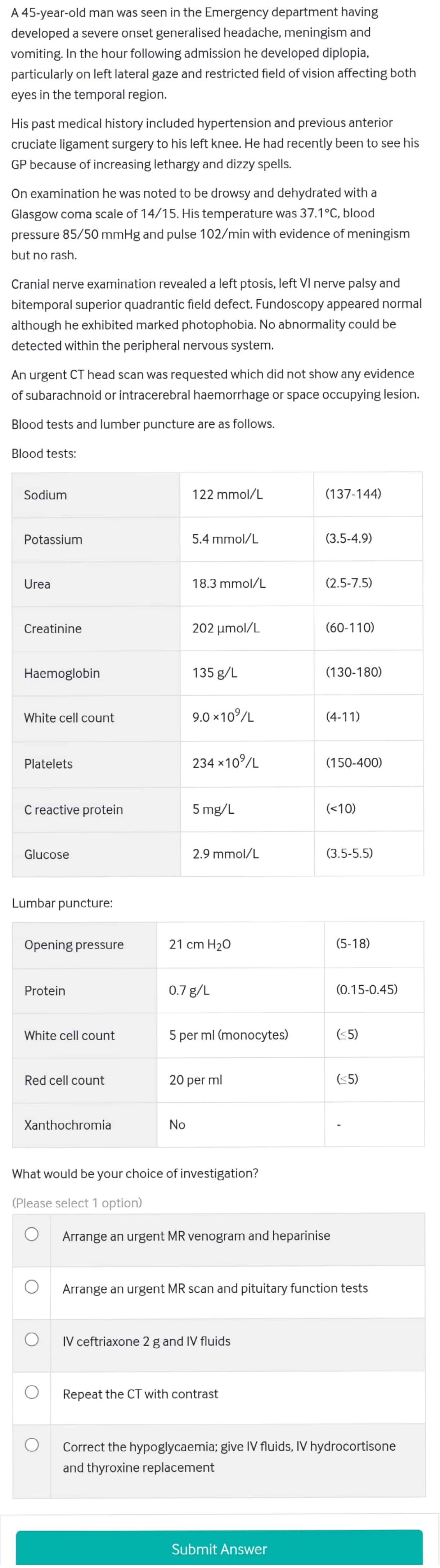
The diagnosis is made by duodenal biopsy demonstrating PAS positive foamy macrophages throughout the lamina propria and the bacillus *Tropheryma whippelii*. It is a rare condition and the means of transmission is unknown.

Other multi-system disorders need to be considered such as sarcoidosis or tuberculosis. However given a normal CXR and normal CSF analysis these two diagnoses are unlikely.

New variant Creutzfeld-Jakob disease has a longer duration than sporadic Creutzfeld-Jakob disease and often presents initially with psychiatric symptoms and sensory disturbance.

There is nothing in the history to make one suspicious of HIV, however this would be a differential diagnosis for patients presenting with focal CNS signs and gastrointestinal disturbance.

Next Question



A 45-year-old man was seen in the Emergency department having developed a severe onset generalised headache, meningism and vomiting. In the hour following admission he developed diplopia, particularly on left lateral gaze and restricted field of vision affecting both eyes in the temporal region.

His past medical history included hypertension and previous anterior cruciate ligament surgery to his left knee. He had recently been to see his GP because of increasing lethargy and dizzy spells.

On examination he was noted to be drowsy and dehydrated with a Glasgow coma scale of 14/15. His temperature was 37.1°C, blood pressure 85/50 mmHg and pulse 102/min with evidence of meningism but no rash.

Cranial nerve examination revealed a left ptosis, left VI nerve palsy and bitemporal superior quadrantic field defect. Fundoscopy appeared normal although he exhibited marked photophobia. No abnormality could be detected within the peripheral nervous system.

An urgent CT head scan was requested which did not show any evidence of subarachnoid or intracerebral haemorrhage or space occupying lesion.

Blood tests and lumber puncture are as follows.

Blood tests:

Sodium	122 mmol/L	(137-144)
Potassium	5.4 mmol/L	(3.5-4.9)
Urea	18.3 mmol/L	(2.5-7.5)
Creatinine	202 µmol/L	(60-110)
Haemoglobin	135 g/L	(130-180)
White cell count	9.0×10^9/L	(4-11)
Platelets	234×10^9/L	(150-400)
C reactive protein	5 mg/L	(<10)
Glucose	2.9 mmol/L	(3.5-5.5)

Lumbar puncture:

Opening pressure	21 cm H_2O	(5-18)
Protein	0.7 g/L	(0.15-0.45)
White cell count	5 per ml (monocytes)	(≤5)
Red cell count	20 per ml	(≤5)
Xanthochromia	No	-

What would be your choice of investigation?

(Please select 1 option)

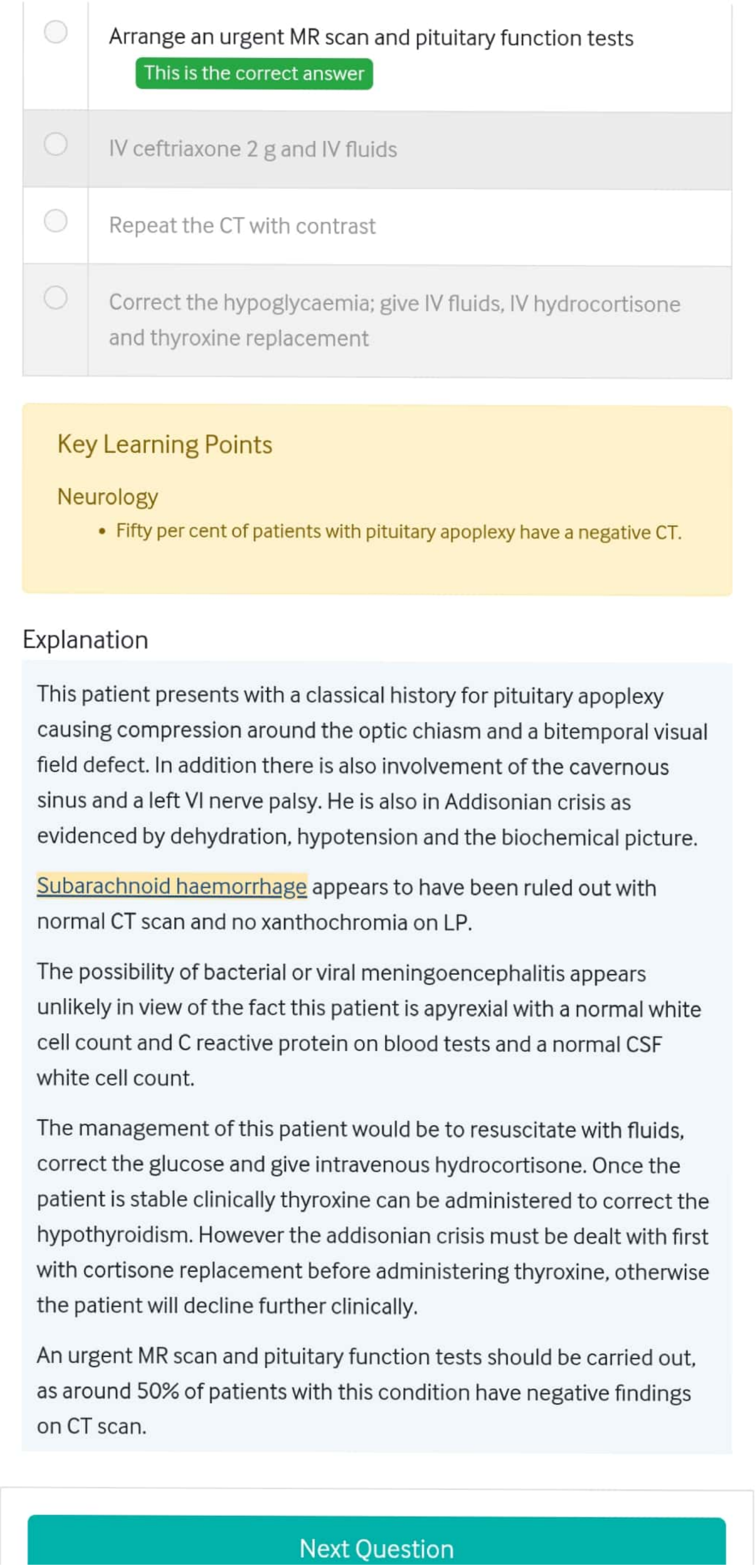
○ Arrange an urgent MR venogram and heparinise

○ Arrange an urgent MR scan and pituitary function tests

○ IV ceftriaxone 2 g and IV fluids

○ Repeat the CT with contrast

○ Correct the hypoglycaemia; give IV fluids, IV hydrocortisone and thyroxine replacement



○ Arrange an urgent MR scan and pituitary function tests

This is the correct answer

○ IV ceftriaxone 2 g and IV fluids

○ Repeat the CT with contrast

○ Correct the hypoglycaemia; give IV fluids, IV hydrocortisone and thyroxine replacement

Key Learning Points

Neurology

- Fifty per cent of patients with pituitary apoplexy have a negative CT.

Explanation

This patient presents with a classical history for pituitary apoplexy causing compression around the optic chiasm and a bitemporal visual field defect. In addition there is also involvement of the cavernous sinus and a left VI nerve palsy. He is also in Addisonian crisis as evidenced by dehydration, hypotension and the biochemical picture.

Subarachnoid haemorrhage appears to have been ruled out with normal CT scan and no xanthochromia on LP.

The possibility of bacterial or viral meningoencephalitis appears unlikely in view of the fact this patient is apyrexial with a normal white cell count and C reactive protein on blood tests and a normal CSF white cell count.

The management of this patient would be to resuscitate with fluids, correct the glucose and give intravenous hydrocortisone. Once the patient is stable clinically thyroxine can be administered to correct the hypothyroidism. However the addisonian crisis must be dealt with first with cortisone replacement before administering thyroxine, otherwise the patient will decline further clinically.

An urgent MR scan and pituitary function tests should be carried out, as around 50% of patients with this condition have negative findings on CT scan.

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A 60-year-old man presented to the Emergency department having developed weakness and excruciating pain of his right lower limb. The weakness and pain had progressively worsened over several weeks to the point where he was wheelchair bound. The pain was deep-seated and confined to the right groin. He did not complain of any back pain, but had noticed increased urinary frequency and hesitancy.

He had a history of diabetes and hypertension, and took regular metformin and ramipril. He was a smoker of 20 cigarettes per day and drank six units of alcohol per week.

On examination he appeared in some distress. There was evidence of right leg oedema and the leg itself appeared warm and dry to touch. Tone was diminished at the hip, knee, ankle and foot and there was evidence of wasting over the anterior and posterior compartments of the right lower leg. There was 3/5 power of hip flexion, extension and adduction with marked weakness of knee flexion, dorsiflexion, plantar flexion, eversion and inversion. The knee, ankle and plantar reflexes were all absent. On testing sensation, there was diminished pinprick over the toes, extending to the perianal area in the right leg. The left leg appeared normal.

Investigations revealed:

Haemoglobin	125 g/L	(130-180)
White cell count	11.1 $\times 10^9$/L	(4-11)
Platelets	345 $\times 10^9$/L	(150-400)
Serum sodium	134 mmol/L	(137-144)
Serum potassium	3.5 mmol/L	(3.5-4.9)
Serum urea	6.7 mmol/L	(2.5-7.5)
Serum creatinine	123 µmol/L	(60-110)
Serum corrected calcium	3.1 mmol/L	(2.2-2.6)

What is the most likely diagnosis?

(Please select 1 option)

- ○ Lumbosacral plexopathy
- ○ Common peroneal neuropathy
- ○ Femoral neuropathy
- ○ S1 radiculopathy
- ○ Sciatic nerve palsy

Submit Answer

What is the most likely diagnosis?

(Please select 1 option)

○ Lumbosacral plexopathy **This is the correct answer**

○ Common peroneal neuropathy

○ Femoral neuropathy

○ S1 radiculopathy

● Sciatic nerve palsy **Incorrect answer selected**

Key Learning Points

Neurology

- Cancer and metastatic spread are a recognised cause of plexopathy.

Explanation

This patient presents with generalised weakness of the right leg associated with pelvic pain, leg oedema and autonomic dysfunction. The most likely diagnosis is a lumbosacral plexopathy.

There is a suggestion in the history of possible prostate dysfunction with urinary frequency and hesitancy and also, there is hypercalcaemia found on biochemical testing. This suggests a possible metastatic cause for the plexopathy.

Upper lumbar plexus lesion will cause weakness of hip flexion and adduction of the thigh and extension of the leg with anaesthesia over the anterior thigh and leg. Lower plexus lesions will weaken the posterior thigh and foot muscles. Lesions affecting the entire plexus will affect all muscle groups causing weakness or paralysis of the leg, areflexia and anaesthesia from the toes, to involve the perianal area.

The involvement of thigh adduction, flexion and anaesthesia over the anterior thigh would go against a sciatic nerve palsy (L4-S3), which supplies the hamstrings and all muscles below the knee.

A femoral neuropathy (L2-4) would help explain hip weakness, knee extension and anaesthesia over the anterior thigh, but would not explain weakness of the foot.

A common peroneal nerve palsy would cause weakness of dorsiflexion and eversion with preserved inversion.

A 64-year-old retired librarian presented to her general practitioner with progressive unsteadiness of gait resulting in her having to furniture walk around her house.

Recently she had noticed great difficulty in reading her books and frequently lost her place whilst scanning the text. Her husband, who accompanied her to clinic, had noticed a slowing of performing simple tasks such as making a meal, washing or eating and now needed to accompany her to the shops and help in most tasks because of her unsteadiness.

Her past medical history included hypertension, hypothyroidism and osteoarthritis. Her regular medication included bendroflumethiazide 2.5 mg daily and thyroxine 75 mcg daily.

On examination her blood pressure was 102/65 mmHg (lying) and 97/55 mmHg (standing). Her pulse was 67/minute and regular and she had marked dysarthria. On examination of eye movements there was normal smooth pursuit, but slow vertical saccades and evidence of square wave jerks. She also had slow spastic tongue movements. On examination of the upper limb, there was marked neck rigidity with retrocollis and evidence of symmetrically increased tone and bradykinesia. On examination of the lower limb, there was symmetrically increased tone, bradykinesia and gait instability.

Investigations revealed:

Serum sodium	135 mmol/L	(137 - 144)
Serum potassium	3.8 mmol/L	(3.5 - 4.9)
Serum urea	5.4 mmol/L	(2.5 - 7.5)
Serum creatinine	100 mol/L	(60 - 110)
Serum thyroxine	60 nmol/L	(58 - 178)
Serum thyroid stimulating hormone	6.5 mU/L	(0.4 - 5)

An MRI scan of her brain showed midbrain atrophy.

What is the likely cause for this patient's symptoms and signs?

(Please select 1 option)

○ Parkinson’s disease

○ Progressive supranuclear palsy

○ Cerebrovascular disease

○ Multiple systems atrophy

○ Hypothyroidism

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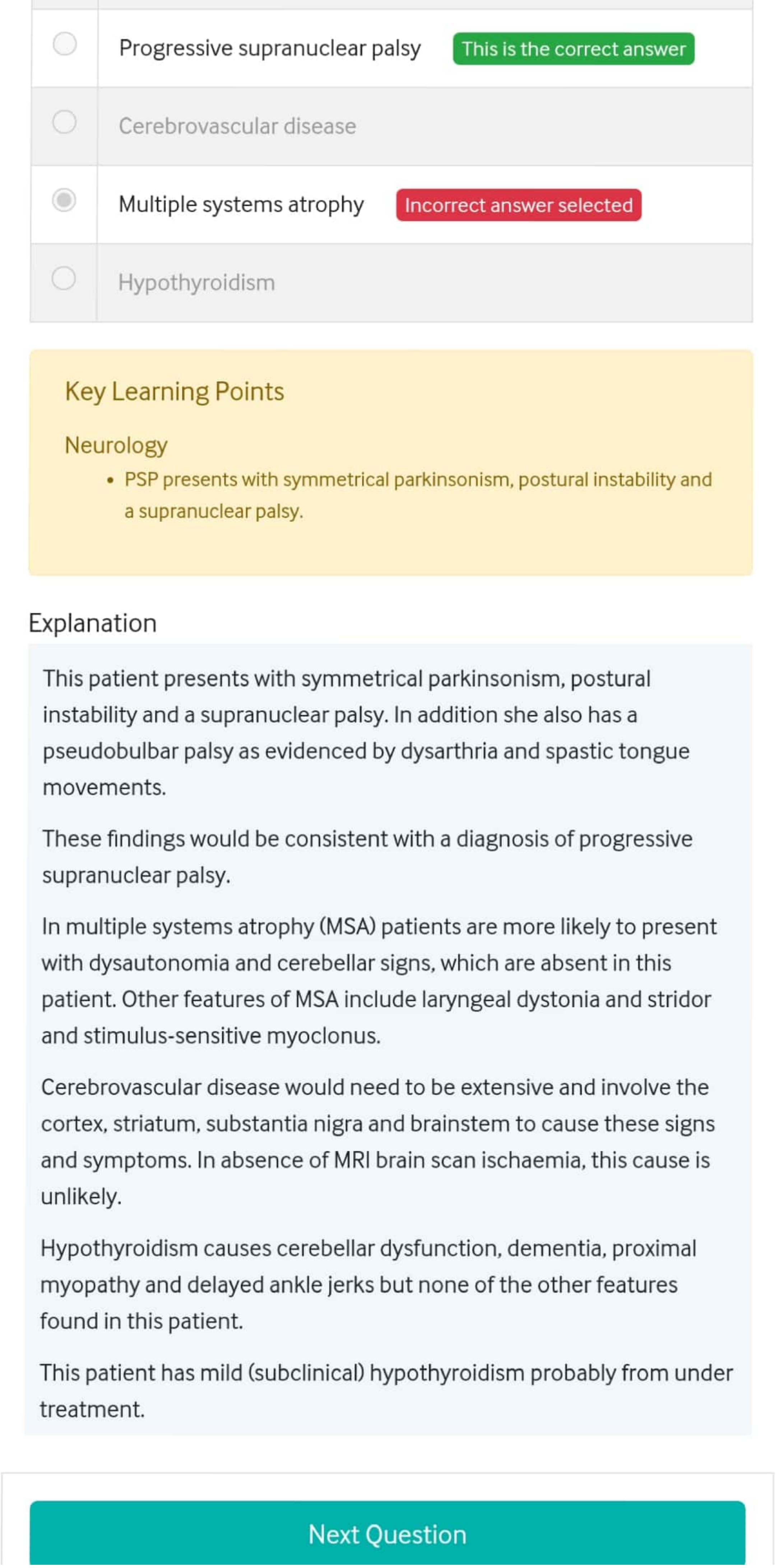

○ Progressive supranuclear palsy

○ Cerebrovascular disease

● Multiple systems atrophy

○ Hypothyroidism

Key Learning Points

Neurology

- PSP presents with symmetrical parkinsonism, postural instability and a supranuclear palsy.

Explanation

This patient presents with symmetrical parkinsonism, postural instability and a supranuclear palsy. In addition she also has a pseudobulbar palsy as evidenced by dysarthria and spastic tongue movements.

These findings would be consistent with a diagnosis of progressive supranuclear palsy.

In multiple systems atrophy (MSA) patients are more likely to present with dysautonomia and cerebellar signs, which are absent in this patient. Other features of MSA include laryngeal dystonia and stridor and stimulus-sensitive myoclonus.

Cerebrovascular disease would need to be extensive and involve the cortex, striatum, substantia nigra and brainstem to cause these signs and symptoms. In absence of MRI brain scan ischaemia, this cause is unlikely.

Hypothyroidism causes cerebellar dysfunction, dementia, proximal myopathy and delayed ankle jerks but none of the other features found in this patient.

This patient has mild (subclinical) hypothyroidism probably from under treatment.

A 59-year-old builder was referred to the neurology clinic with a four month history of weakness and clumsiness affecting the right arm and hand.

He had noticed significantly reduced dexterity in the right hand and fingers and weakness of grip to the point where he had had to give up his building job. He had not noticed any reduced sensation in the right hand or forearm. He had a past medical history of asthma, osteoarthritis affecting the hands and knees and hypertension. He was a smoker of 10 cigarettes per day and drank 21 units of alcohol per week.

On examination he was of slim build. Upper limb examination revealed mild wasting affecting the right dorsal interossei, and right thenar muscles. Fasciculations were also detected. Tone was reduced in the right arm and power was significantly reduced on testing right finger flexion/extension, thumb abduction and opposition and wrist flexion/extension. Shoulder abduction and adduction appeared intact. Reflexes were asymmetrically depressed on the right, however sensation appeared intact in both arms. Lower limb examination appeared normal. Chest and abdominal examination were both normal.

The results of are lumbar puncture:

CSF protein	0.37 g/L	(0.15-0.45)
CSF white cell count	4 cells per ml	(≤5)
CSF red cell count	1 cell per ml	(≤5)
CSF glucose	3.8 mmol/L	(3.3-4.4)

MR Cervical spine shows degenerative joint disease affecting C3-6 vertebrae.

EMG shows fasciculations affecting dorsal and palmar interossei. No evidence of fasciculations detected proximally or in the left arm.

Nerve conduction studies show patchy block of motor nerve conduction innervating fasciculating muscles Sensory conduction was normal.

What is the likely diagnosis in this patient?

(Please select 1 option)

- ○ Multifocal motor neuropathy (MMN)
- ○ Right ulnar nerve palsy
- ○ Motor neurone disease
- ○ Cervical myelopathy secondary to degenerative joint disease
- ○ Chronic inflammatory demyelinating neuropathy

Submit Answer

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● Multifocal motor neuropathy (MMN) **Correct**

○ Right ulnar nerve palsy

○ Motor neurone disease

○ Cervical myelopathy secondary to degenerative joint disease

○ Chronic inflammatory demyelinating neuropathy

Key Learning Points

Neurology

- MMN is an autoimmune neuropathy associated in 80% of cases with an elevated anti-GM1 ganglioside antibody.

Explanation

This patient presents with marked asymmetrical lower motor neurone weakness affecting the right arm/forearm, in absence of sensory findings, with weakness disproportionate to the degree of wasting. These findings are in keeping with a diagnosis of multifocal motor neuropathy (MMN).

MMN is an autoimmune neuropathy associated in 80% of cases with an elevated anti-GM1 ganglioside antibody and typically presents with progressive weakness affecting an isolated limb without sensory involvement.

Rarely there is cranial nerve or respiratory involvement. There is persistent motor conduction block of the innervating nerves and depressed reflexes as evidenced by clinical examination and paraclinical evidence.

It is an important differential diagnosis of motor neurone disease as it is potentially treatable. Treatment with IV immunoglobulin may be very effective in some patients. Cyclophosphamide is also used on a long term basis.

In chronic inflammatory demyelinating neuropathy (CIDP), the cerebrospinal fluid (CSF) protein is elevated in addition to the clinical findings.

In motor neurone disease, generalised fasciculations would have been evident on EMG and not confined to the wasted muscles.

A cervical myelopathy would cause upper motor neurone signs in the lower limbs and may lead to lower motor signs or hypo-reflexia in the upper limb.

Right ulnar nerve palsy would account for weakness of the small muscles in the hand, but would not account for thenar and forearm muscle involvement.

Next Question

Question 82

Question 82 of 133

< >

A 35-year-old man presents to first fit clinic.

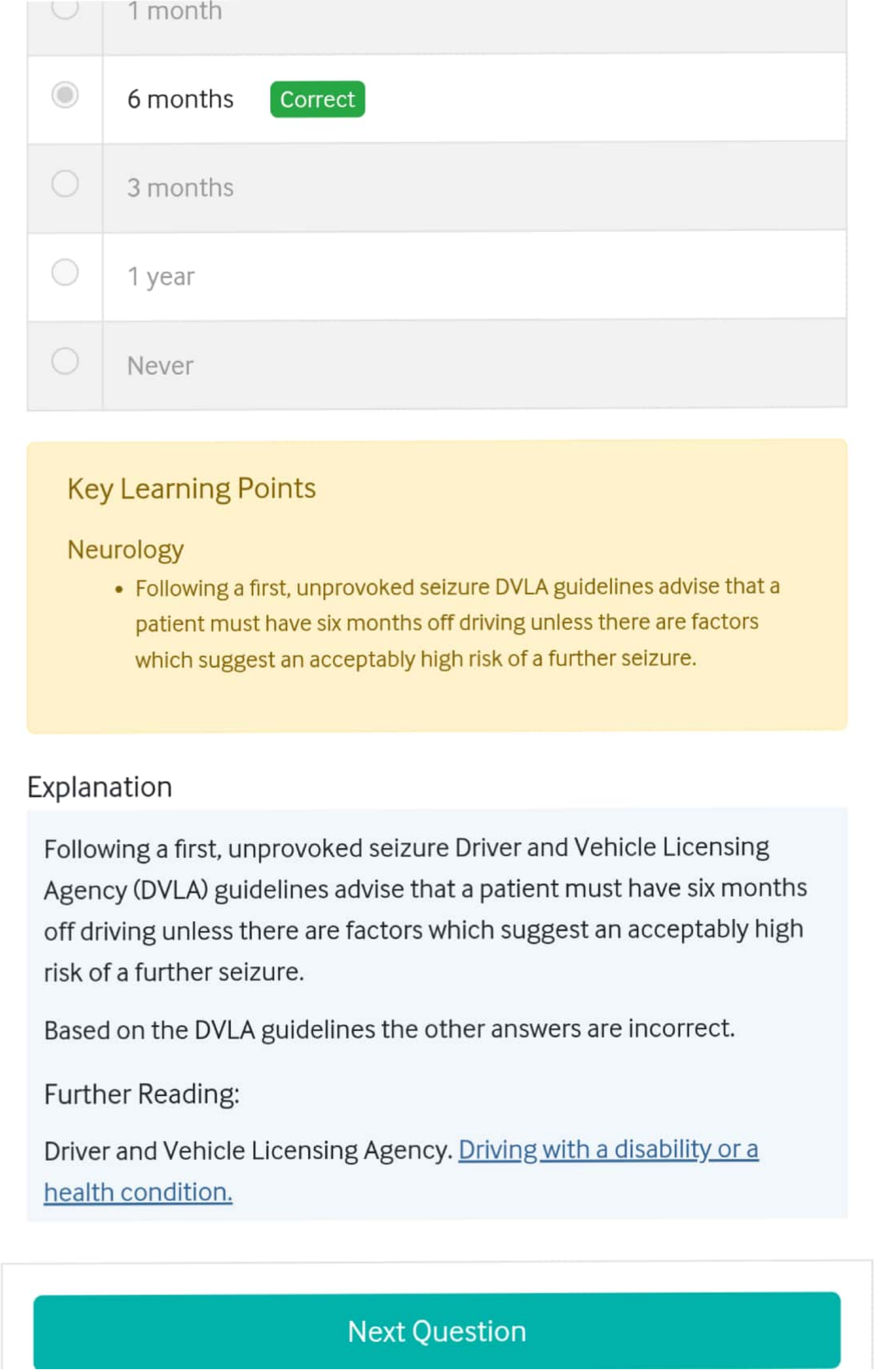
He had a witnessed single tonic clonic seizure lasting a couple of minutes. An MRI and EEG were performed and were reported as normal. He was not commenced on any treatment and was advised not to drive when he was discharged from hospital.

If he remains seizure free when is he able to recommence driving?

(Please select 1 option)

- ○ 1 month
- ○ 6 months
- ○ 3 months
- ○ 1 year
- ○ Never

Submit Answer



○ 1 month

● 6 months **Correct**

○ 3 months

○ 1 year

○ Never

Key Learning Points

Neurology

- Following a first, unprovoked seizure DVLA guidelines advise that a patient must have six months off driving unless there are factors which suggest an acceptably high risk of a further seizure.

Explanation

Following a first, unprovoked seizure Driver and Vehicle Licensing Agency (DVLA) guidelines advise that a patient must have six months off driving unless there are factors which suggest an acceptably high risk of a further seizure.

Based on the DVLA guidelines the other answers are incorrect.

Further Reading:

Driver and Vehicle Licensing Agency. Driving with a disability or a health condition.

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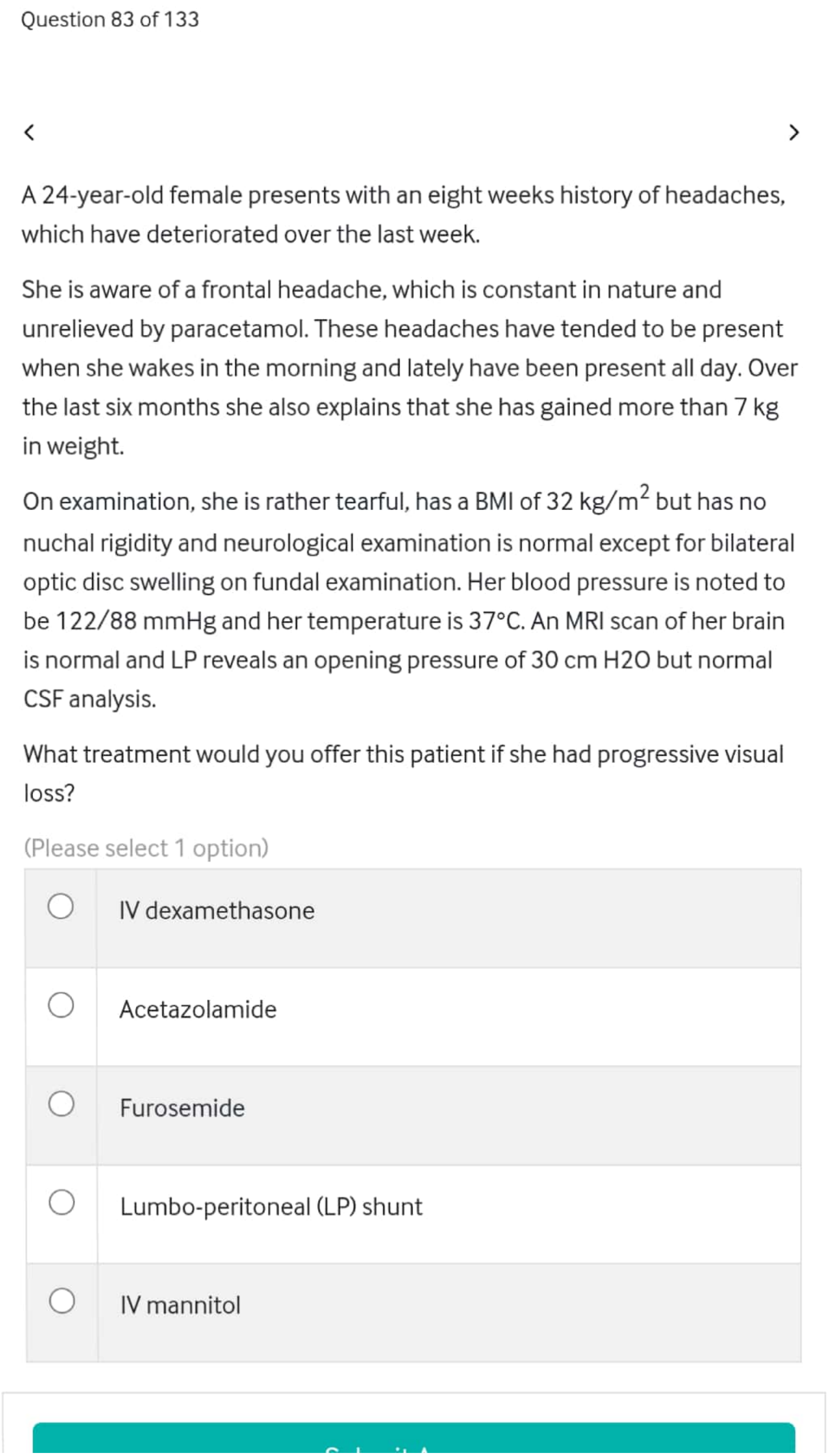

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A 24-year-old female presents with an eight weeks history of headaches, which have deteriorated over the last week.

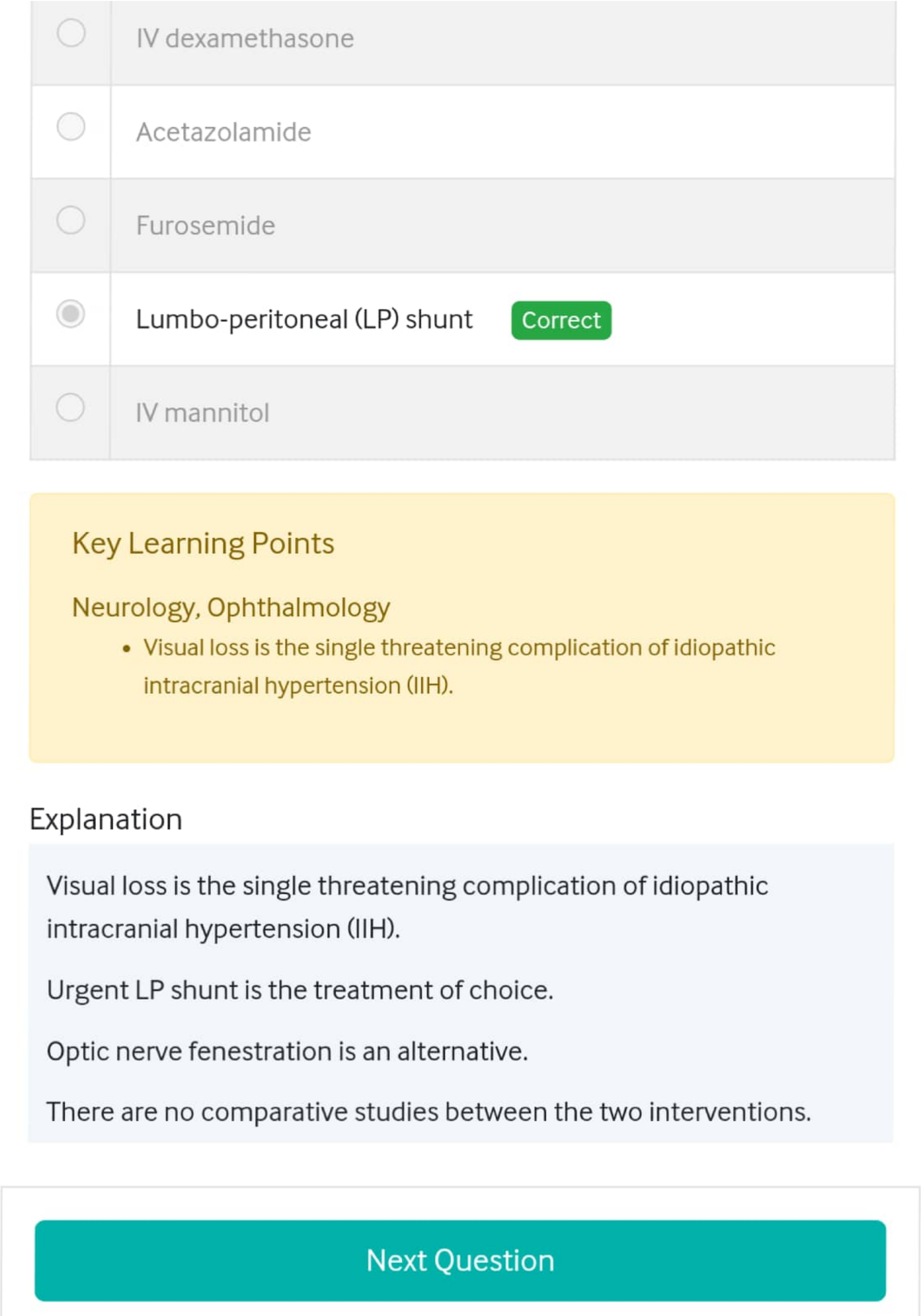
She is aware of a frontal headache, which is constant in nature and unrelieved by paracetamol. These headaches have tended to be present when she wakes in the morning and lately have been present all day. Over the last six months she also explains that she has gained more than 7 kg in weight.

On examination, she is rather tearful, has a BMI of 32 kg/m^2 but has no nuchal rigidity and neurological examination is normal except for bilateral optic disc swelling on fundal examination. Her blood pressure is noted to be 122/88 mmHg and her temperature is 37°C. An MRI scan of her brain is normal and LP reveals an opening pressure of 30 cm H2O but normal CSF analysis.

What treatment would you offer this patient if she had progressive visual loss?

(Please select 1 option)

○	IV dexamethasone
○	Acetazolamide
○	Furosemide
○	Lumbo-peritoneal (LP) shunt
○	IV mannitol



○ IV dexamethasone

○ Acetazolamide

○ Furosemide

● Lumbo-peritoneal (LP) shunt **Correct**

○ IV mannitol

Key Learning Points

Neurology, Ophthalmology

- Visual loss is the single threatening complication of idiopathic intracranial hypertension (IIH).

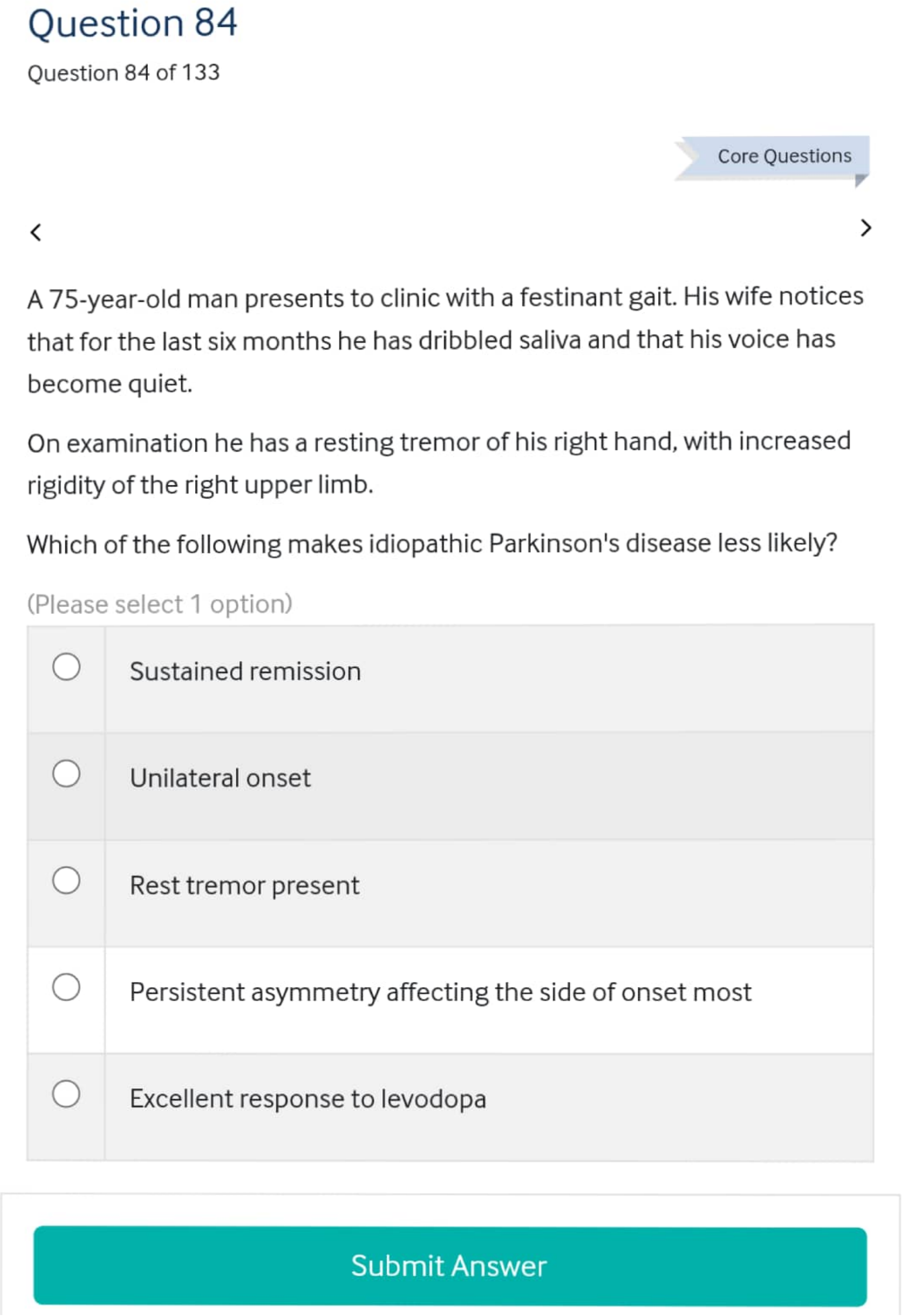
Explanation

Visual loss is the single threatening complication of idiopathic intracranial hypertension (IIH).

Urgent LP shunt is the treatment of choice.

Optic nerve fenestration is an alternative.

There are no comparative studies between the two interventions.



Question 84

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A 75-year-old man presents to clinic with a festinant gait. His wife notices that for the last six months he has dribbled saliva and that his voice has become quiet.

On examination he has a resting tremor of his right hand, with increased rigidity of the right upper limb.

Which of the following makes idiopathic Parkinson's disease less likely?

(Please select 1 option)

○ Sustained remission

○ Unilateral onset

○ Rest tremor present

○ Persistent asymmetry affecting the side of onset most

○ Excellent response to levodopa

(Please select 1 option)

○ Sustained remission **This is the correct answer**

○ Unilateral onset

○ Rest tremor present

● Persistent asymmetry affecting the side of onset most **Incorrect answer selected**

○ Excellent response to levodopa

Key Learning Points

Neurology

- Parkinson's disease is a progressive disorder and evidence of sustained remission excludes the disease.

Explanation

According to the UKPDS Brain Bank criteria diagnosis of a parkinsonian syndrome is made in the presence of bradykinesia plus one of the following of

- Muscular rigidity
- Rest tremor
- Postural instability.

Parkinson's disease is a progressive disorder and evidence of sustained remission excludes the disease.

All other options are considered supportive criteria in the diagnosis of Parkinson's disease and three or more are required for a diagnosis of definite Parkinson's disease.

<   >

A 30-year-old woman presented with a six month history of numbness and tingling over the left side of her face, and the right arm and leg. She denied any weakness of the arms or legs and did not complain of any bladder disturbances, headaches or visual problems.

The problem appeared to be intermittent. She had a past medical history of irritable bowel syndrome and chronic lower back pain. She was unemployed, smoked 30 cigarettes per day and drank 20 units of alcohol per week. She lived at home with three children aged 9, 5 and 3 years. She admitted that she occasionally used cocaine.

On examination, she was alert and orientated. On cranial nerve examination there was decreased pinprick sensation affecting the whole of the left side of her face including the angle of jaw. Scalp sensation was normal. The rest of the cranial nerves were normal. On peripheral nerve examination there was impaired pinprick and vibration sensation affecting the whole of the right arm and leg. Tone, power, reflexes and coordination were all normal.

An MRI scan of the brain with gadolinium contrast was normal. A lumbar puncture was performed and the following data obtained:

Opening pressure	130 mm H_2O	(50-180)
CSF protein	0.45 g/L	(0.15-0.45)
CSF white cell count	5 cells per ml	(≤5)
CSF red cell count	2 cells per ml	(≤5)
CSF oligoclonal bands	Positive	
Serum oligoclonal bands	Positive	

What is the most likely diagnosis?

(Please select 1 option)

○ Functional disorder

○ Multiple sclerosis

○ Brainstem infarct secondary to cocaine

○ Parietal lobe seizures

○ Vitamin B12 deficiency

Submit Answer

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(Please select 1 option)

● Functional disorder **Correct**

○ Multiple sclerosis

○ Brainstem infarct secondary to cocaine

○ Parietal lobe seizures

○ Vitamin B12 deficiency

Key Learning Points

Neurology

- Oligoclonal bands which are present in the cerebrospinal fluid and matched in the serum is a non-specific biochemical finding.

Explanation

This patient presents with a six month history of intermittent right sided arm and leg sensory disturbance and left sided facial sensory disturbance. This places the likely cause of the problem within the brainstem.

The sensory examination although quite subjective, is peculiar in that the angle of the jaw is affected (C2) yet scalp sensation back to the vertex is not affected (V). The MRI imaging is entirely normal, so the likelihood of demyelination is very small.

Secondly although oligoclonal bands are present in the cerebrospinal fluid, they are matched in the serum, which is non-specific.

Brainstem infarct secondary to cocaine use would have presented as an acute onset of cranial dysfunction together with long tract signs.

Vitamin B12 deficiency can present as a neuropathy or subacute combined degeneration of the cord. There are no other features in the history to suggest this diagnosis.

Parietal lobe seizures can present with a variety of sensory disturbances, however one would expect contralateral face and limb paraesthesia, which may spread in a jacksonian manner.

Next Question

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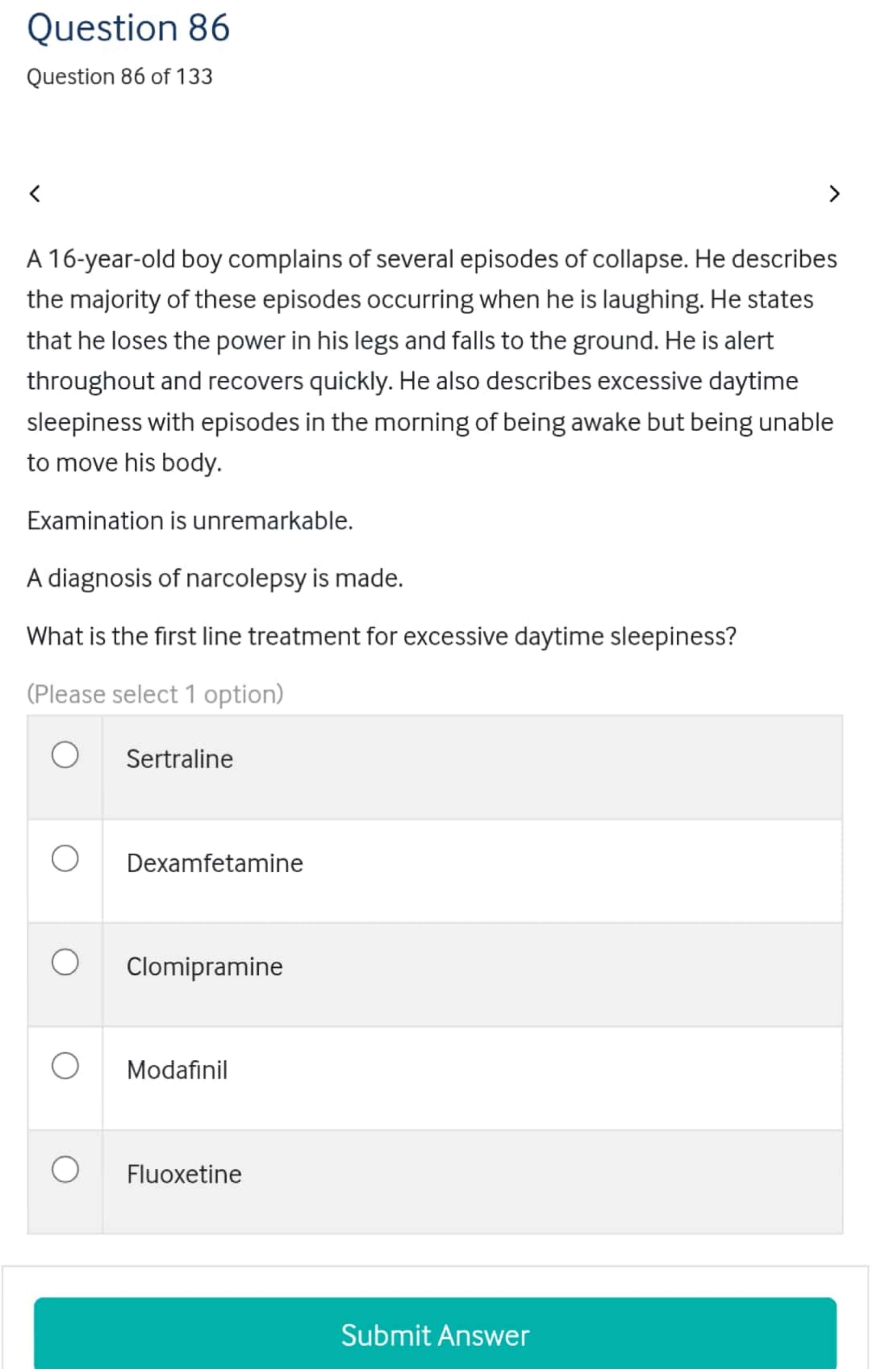

Question 86

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A 16-year-old boy complains of several episodes of collapse. He describes the majority of these episodes occurring when he is laughing. He states that he loses the power in his legs and falls to the ground. He is alert throughout and recovers quickly. He also describes excessive daytime sleepiness with episodes in the morning of being awake but being unable to move his body.

Examination is unremarkable.

A diagnosis of narcolepsy is made.

What is the first line treatment for excessive daytime sleepiness?

(Please select 1 option)

- ○ Sertraline
- ○ Dexamfetamine
- ○ Clomipramine
- ○ Modafinil
- ○ Fluoxetine

○ Clomipramine

● Modafinil **Correct**

○ Fluoxetine

Key Learning Points

Neurology

- modafinil is first line for the treatment of excessive daytime sleepiness.

Explanation

Narcolepsy is a rare condition which is characterised by excessive daytime sleepiness, sleep paralysis, hypnagogic hallucinations, and cataplexy (sudden collapse triggered by emotion such as laughing or crying).

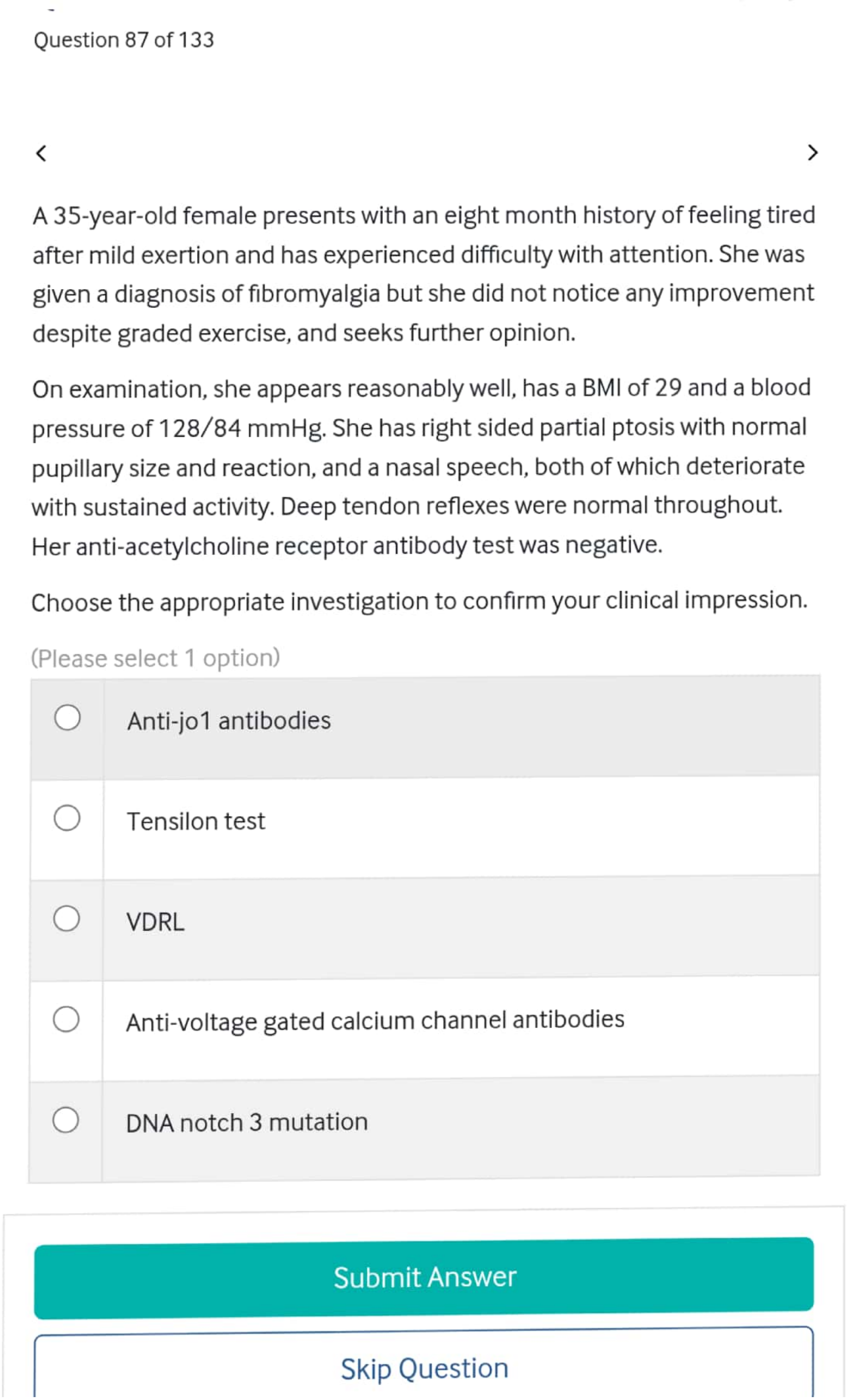
In 1999 modafinil was approved for the treatment of excessive daytime sleepiness.

There is no cure for narcolepsy.

Clomipramine, fluoxetine and sertraline are used in the management of cataplexy.

Amphetamine based compounds such as dexamphetamine have been used to control excessive sleepiness but are used less commonly due to risks of addiction.

Next Question



Question 87 of 133

A 35-year-old female presents with an eight month history of feeling tired after mild exertion and has experienced difficulty with attention. She was given a diagnosis of fibromyalgia but she did not notice any improvement despite graded exercise, and seeks further opinion.

On examination, she appears reasonably well, has a BMI of 29 and a blood pressure of 128/84 mmHg. She has right sided partial ptosis with normal pupillary size and reaction, and a nasal speech, both of which deteriorate with sustained activity. Deep tendon reflexes were normal throughout. Her anti-acetylcholine receptor antibody test was negative.

Choose the appropriate investigation to confirm your clinical impression.

(Please select 1 option)

- ○ Anti-jo1 antibodies
- ○ Tensilon test
- ○ VDRL
- ○ Anti-voltage gated calcium channel antibodies
- ○ DNA notch 3 mutation

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● Tensilon test **Correct**

○ VDRL

○ Anti-voltage gated calcium channel antibodies

○ DNA notch 3 mutation

Key Learning Points

Neurology

- Myasthenia Gravis can present with negative acetyl choline receptor antibodies.

Explanation

This patient is likely to have acetylcholine receptor (AChR) Ab negative myasthenia gravis.

The differential would also include congenital myasthenic syndromes and Eaton-Lambert syndrome. The former are unlikely given their rarity and the patient's age. Similarly in Eaton-Lambert syndrome eye signs are unusual.

ACh receptor antibodies are negative in 15% of generalised myasthenia gravis; the picture is suggestive of myasthenia gravis (which is much more common than the other two possibilities), so doing a tensilon test and repetitive (not conventional) electromyogram (EMG) are the most appropriate choices here.

Next Question

A 47-year-old gentleman was seen in neurology clinic complaining of a five day history of 'flu-like symptoms' and double vision.

The diplopia occurred on looking to the right and also whilst reading a book. In the last two days he had been unable to close his right eye fully and had also noticed a similar problem with the left eye. He had recently been diagnosed as having type 2 diabetes mellitus by his GP and had been started on a diabetic diet.

He had no other past medical history and did not take any regular medication. He smoked 10 cigarettes per day but lead a reasonably active lifestyle and had recently returned from a walking holiday in New England, USA with his wife.

On examination he was alert and orientated. His temperature was 37.3°C, blood pressure was 146/82 mmHg, pulse 85/minute and blood glucose monitoring level 5.4. There was no rash. Fundoscopy was normal, and pupils were equal and reactive to light. There was oculoparesis affecting the right lateral rectus and also the right superior oblique muscle. There was also evidence of bilateral facial weakness and bilateral Bell's phenomena. Sensory examination also revealed an area of anaesthesia over the right cheek and there was an absent right corneal reflex.

The peripheral nervous system examination was normal, however he complained of arthralgia in his hands and knees.

Results of lumbar puncture:

Opening pressure	10 cm H_2O	(8-15)
CSF Protein	1.15 g/L	(0.15-0.45)
CSF white cell count	120 per ml (80% lymphocytes)	(0-5)
CSF red cell count	4 per ml	(<5)
CSF glucose	3.6 mmol/L	(3.3-4.4)
CSF Oligoclonal bands	Present	-
Plasma glucose	5.5 mmol/L	(3.0-6.0)
Serum oligoclonal bands	Negative	-

MRI brain scan shows punctate periventricular white matter lesions.

What is the likely diagnosis?

(Please select 1 option)

○ Pontine lesion

○ Multiple sclerosis

○ Diabetic-related mononeuritis multiplex

○ Guillain-Barré syndrome

○ Neuroborreliosis

Submit Answer

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○	Guillain-Barre syndrome
●	Neuroborreliosis Correct

Key Learning Points

Neurology

- Patients with multiple nerve palsies who have recently been in an endemic area on holiday should prompt investigation for Neuroborreliosis (Lyme disease).

Explanation

This gentleman presents with a mononeuritis multiplex involving right IV, V, VI, VII and left VII nerve palsies.

He also has flu-like symptoms and arthralgia. Investigations reveal a cerebrospinal fluid (CSF) lymphocytic pleocytosis with intrathecal oligoclonal band production. His MRI scan would be in keeping with a demyelinating or inflammatory disorder. Neuroborreliosis (Lyme disease) should be suspected, given the patient has recently been in an endemic area on holiday.

The early phase of the illness is characterised by an expanding rash (erythema migrans) caused by a tick bite, however three to four weeks post bite the rash has faded, and often the patient is unaware of having been bitten.

Multiple sclerosis is a central demyelinating condition often presenting with an optic neuritis.

With a brainstem pontine lesion, one would expect pyramidal signs in the contralateral upper/lower limb.

A pontine lesion would not explain a fourth cranial nerve palsy.

Guillain-Barré syndrome can be associated with cranial nerve palsies but most patients present with a sensory-motor demyelinating polyradiculoneuropathy and would be areflexic.

Diagnosis is by *Borrelia burgdorferi* serological testing with enzyme-linked immunosorbent assay (ELISA) or immunofluorescence assay (IFA) having good sensitivity (89%) but lower specificity (72%) in detecting patients with Lyme disease.

Next Question

A 30-year-old woman was referred to first fit clinic, having presented to the Emergency department three weeks ago with a seizure.

The seizure occurred at approximately 3 am and was witnessed by her boyfriend. He gave an account whereby he had woken up to find his girlfriend violently shaking her arms and legs. The shaking lasted approximately three minutes and he commented her breathing was laboured and she had blood in her mouth and had been incontinent of urine. The day before she had celebrated her birthday and had consumed approximately one bottle of champagne. She had felt tired prior to retiring to bed, but had otherwise felt fine.

She had a history of asthma and frequent headaches and took regular sumatriptan and a salbutamol inhaler as required. She was adopted. She was a smoker of 10 cigarettes per day and drank approximately 20 units of alcohol per week. She held a driving licence and was at present driving. She worked in publishing.

Examination revealed a blood pressure of 120/80 mmHg, pulse of 67 beats/minute and normal heart sounds. Examination of the neurological system was entirely normal.

Investigations revealed:

Haemoglobin	121 g/L	(115-165)
Mean cell volume	100 fL	(80-96)
White cell count	6.7 $\times 10^9$/L	(4-11)
Platelets	325 $\times 10^9$/L	(150-400)
Sodium	135 mmol/L	(137-144)
Potassium	4.2 mmol/L	(3.5-4.9)
Urea	4.3 mmol/L	(3.5-4.9)
Creatinine	88 µmol/L	(60-110)
Gamma GT	250 U/L	(0-51)
Alkaline phosphatase	287 U/L	(40-150)
Alanine aminotransferase	67 U/L	(0-50)
Platelets	325 $\times 10^9$/L	(150-400)

Given the above clinical account, how would you next manage this patient?

(Please select 1 option)

○ Commence on sodium valproate and advise not to drive

○ Commence on lamotrigine and advise not to drive

○ Arrange for an EEG and head imaging to incorporate MRI and head CT

○ Advise to cut down on her alcohol intake. She is allowed to continue driving as this was a provoked seizure.

○ Commence on carbamazepine and advise not to drive

Submit Answer

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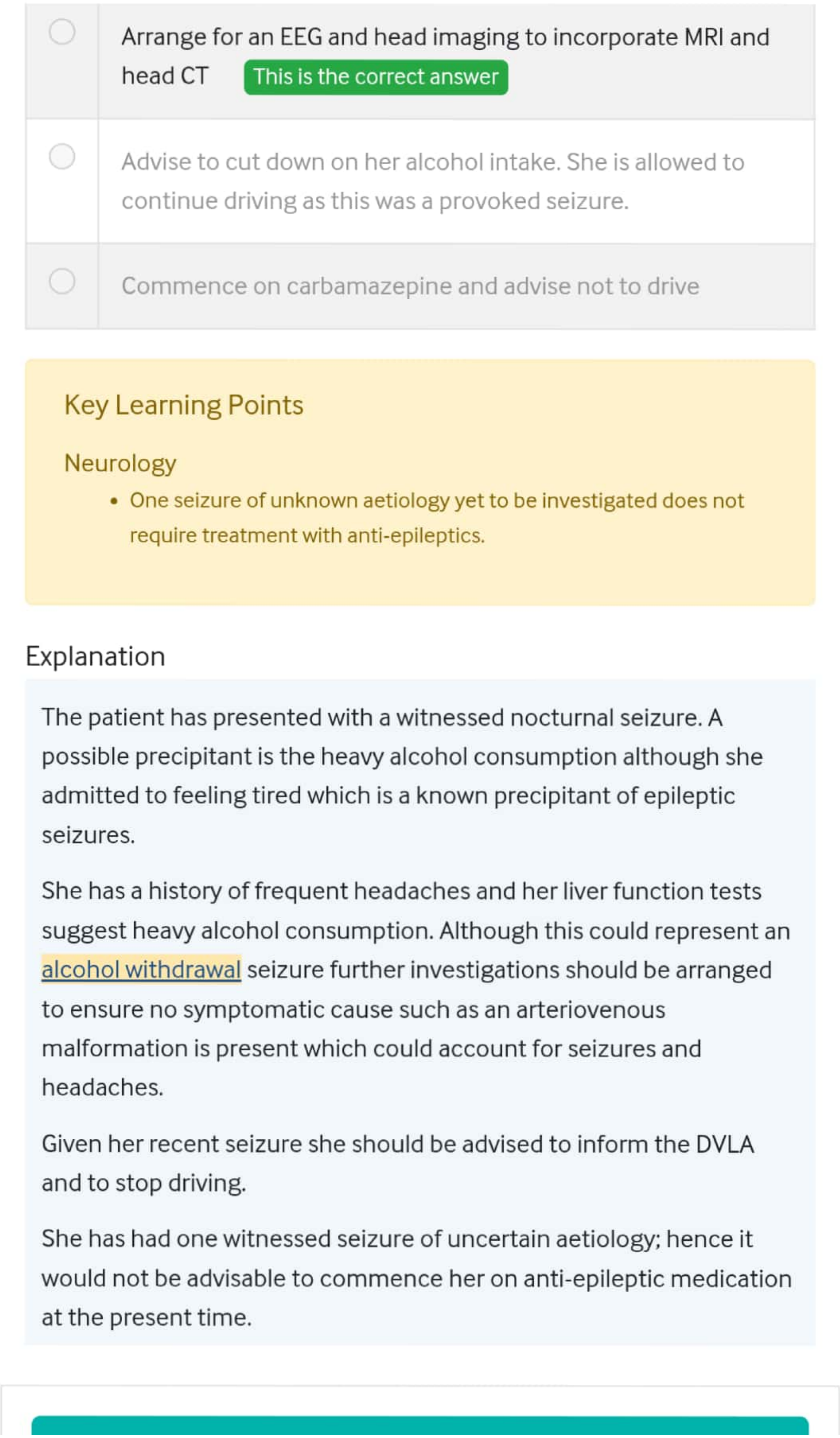

○ Arrange for an EEG and head imaging to incorporate MRI and head CT

○ Advise to cut down on her alcohol intake. She is allowed to continue driving as this was a provoked seizure.

○ Commence on carbamazepine and advise not to drive

Key Learning Points

Neurology

- One seizure of unknown aetiology yet to be investigated does not require treatment with anti-epileptics.

Explanation

The patient has presented with a witnessed nocturnal seizure. A possible precipitant is the heavy alcohol consumption although she admitted to feeling tired which is a known precipitant of epileptic seizures.

She has a history of frequent headaches and her liver function tests suggest heavy alcohol consumption. Although this could represent an alcohol withdrawal seizure further investigations should be arranged to ensure no symptomatic cause such as an arteriovenous malformation is present which could account for seizures and headaches.

Given her recent seizure she should be advised to inform the DVLA and to stop driving.

She has had one witnessed seizure of uncertain aetiology; hence it would not be advisable to commence her on anti-epileptic medication at the present time.

A 17-year-old girl was referred to neurology clinic accompanied by her mother.

She gave a nine month history of episodes of altered consciousness whereby she would momentarily lose contact with her surroundings and her eyelids would start to flutter. Her mother had witnessed multiple episodes at home. In addition her friends had witnessed several at college.

She also complained of multiple shock-like flexion movements affecting her arms. These tended to occur during the morning. Most recently however she had been admitted to hospital following two grand mal seizures lasting approximately ten minutes.

Otherwise she had no medical problems and had normal developmental milestones. She was currently studying for her A levels at college. She was a smoker of five cigarettes/day and drank 10 units of alcohol/week. Her cousin had a history of seizures.

On examination she appeared alert and orientated. Fundoscopy was normal as were the rest of the cranial nerves. Examination of the peripheral nervous system did not reveal any abnormalities other than occasional brief flexion movements of both arms.

Investigations revealed:

Haemoglobin	142 g/L	(115-165)
White cell count	6.3×10^9/L	(4-11)
Platelets	301×10^9/L	(150-400)
Serum sodium	141 mmol/L	(137-144)
Serum potassium	4.1 mmol/L	(3.5-4.9)
Serum urea	5.9 mmol/L	(2.5-7.5)
Serum creatinine	75 µmol/L	(60-110)
Fasting plasma glucose	5.4 mmol/L	(3-6)

An MRI scan of the brain was normal. A lumbar puncture was performed and showed:

CSF opening pressure	12 cmH_2O	(8-15)
CSF protein	0.37 g/L	(0.15-0.45)
CSF white cell count	2 cells per ml	(<5)
CSF red cell count	3 cells per ml	(<5)
CSF lactate	1.5 mmol/L	(1-2)

What is the most likely diagnosis?

(Please select 1 option)

○ Complex partial seizures

○ Juvenile myoclonic epilepsy

○ Lennox-Gastaut syndrome

○ Mitochondrial disorder

○ Neuronal ceroid lipofuscinosis

Submit Answer

○ Complex partial seizures

● Juvenile myoclonic epilepsy **Correct**

○ Lennox-Gastaut syndrome

○ Mitochondrial disorder

○ Neuronal ceroid lipofuscinosis

Key Learning Points

Neurology

- Juvenile myoclonic epilepsy is the most common generalised epileptic disorder in childhood/teenage years.

Explanation

This patient gives a history of typical absences; myoclonus and, more recently, generalised seizures. She has a family history of epilepsy, no developmental delay and has no abnormalities on imaging or blood tests. The most likely diagnosis is therefore juvenile myoclonic epilepsy.

The history is not in keeping with a partial seizure disorder, although atypical absences can occur which are more prolonged and are associated with changes in limb tone.

There are no features suggestive of a mitochondrial disorder such as ataxia, cognitive decline or systemic features (short stature, optic atrophy, diabetes, sensorineural deafness), and the CSF lactate is normal.

Neuronal ceroid lipofuscinosis presents in infancy with progressive myoclonic epilepsy and ataxia.

Lennox-Gastaut syndrome also presents in infancy with multiple seizure types and learning difficulties.

Next Question

A 62-year-old man presented to the Emergency department having developed sudden onset right-sided weakness lasting approximately 20 minutes. The weakness had affected his right arm and face and his speech had become slurred. He had had a similar episode the week before lasting approximately 10 minutes.

He was a smoker of 15 cigarettes/day, and took bendroflumethiazide 2.5 mg/day for hypertension. There was no family history of medical problems.

On examination his blood pressure was 140/98 mmHg, pulse was 65 and irregularly irregular and heart sounds were normal. He had a left carotid bruit.

Examination of the neurological system did not reveal any abnormalities. A CT scan with contrast did not show any abnormalities.

Investigations revealed:

Haemoglobin	141 g/L	(130-180)
White cell count	7.5 $\times 10^9$/L	(4-11)
Platelets	332 $\times 10^9$/L	(150-400)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	3.8 mmol/L	(3.5-4.9)
Serum urea	5.7 mmol/L	(2.5-7.5)
Serum creatinine	89 mol/L	(60 - 110)
Serum cholesterol	7.2 mmol/L	(<5.2)
Fasting plasma glucose	5.4 mmol/L	(3.0-6.0)

Doppler ultrasound of the carotid arteries showed a 75% stenosis of left external carotid artery.

Which of the following options offer the best way of managing this patient?

(Please select 1 option)

- ○ Commence on captopril, warfarin and simvastatin
- ○ Commence on atenolol, aspirin and pravastatin
- ○ Commence on perindopril, warfarin and simvastatin
- ○ Commence on digoxin and arrage echocardiogram
- ○ Commence on simvastatin and aspirin and clopidogrel

○ Commence on perindopril, warfarin and simvastatin

This is the correct answer

○ Commence on digoxin and arrage echocardiogram

○ Commence on simvastatin and aspirin and clopidogrel

Key Learning Points

Neurology

- Upto date trials in stroke managent include CHADS2VASC risk scoring and PROGRESS

Explanation

This patient presents with a history in keeping with two transient ischaemic attacks affecting the left anterior circulation. Using the ABCD Oxfordshire prognostic score for the seven day risk of stroke, he scores 5 points placing his risk of stroke in the next seven days at 12.1% (4.2-20.0). His CHADS2VASC score is high and indicative of oral anticoagulation therapy,

He is noted to be in controlled atrial fibrillation, his blood pressure is elevated despite bendroflumethiazide and he is noted to have hypercholesterolaemia. He has a left carotid bruit, although this is emanating from the external carotid artery (as suggested by carotid Dopplers) and is therefore not significant.

He should be commenced on warfarin in view of his atrial fibrillation; a statin and echocardiography should be arranged. His blood pressure is elevated and an additional agent such as perindopril (PROGRESS trial) should be added to his medication.

The notch3 mutation is found in patients with cerebral autosomal dominant arteriopathy with subcortical infarcts (CADASIL). This presents as recurrent subcortical infarcts associated with pseudo bulbar palsy and dementia in mid-adulthood.

A 74-year-old woman accompanied by her daughter was seen in general clinic with episodes of disorientation.

Her daughter had witnessed several of these episodes whereby one minute she could be having a conversation and then next minute she would start asking 'where am I?' She would keep asking this question, but seemed fully aware of her surroundings, and was able to perform tasks in the house and otherwise seemed fairly attentive. These episodes could go on for several hours. Her daughter was particularly concerned as these episodes seemed to post date the loss of her husband from cancer and she was aware that her mother had taken to drinking a moderate amount of alcohol. She had not lost any weight or complained of poor appetite or disturbed sleep.

She had a past medical history of hypertension and arthritis and took regular bendroflumethiazide and Arthrotec. She was a non-smoker and drank approximately 15 units of alcohol per week.

On examination she seemed fully aware of her surroundings but appeared disorientated in time and place and kept asking 'where am I?' When the physician explained she was in hospital she acknowledged the answer, but within minutes was asking the same question. This continued throughout the consultation.

She was able to comply with physical examination which did not reveal any abnormalities within the cranial nerves or peripheral nervous system. General, cardiovascular and abdominal examinations were all normal.

Investigations revealed:

Full blood count	Normal	-
Biochemistry	Normal	-
Thyroxine	59 nmol/L	(58-174)
Thyroid stimulating hormone	6.9 U/L	(<7)

An MRI scan of the brain showed mild generalised cerebral atrophy.

What is the most likely diagnosis?

(Please select 1 option)

○	Alcohol abuse
○	Transient global amnesia
○	Depression
○	Factitious disorder
○	Temporal lobe seizures

Submit Answer

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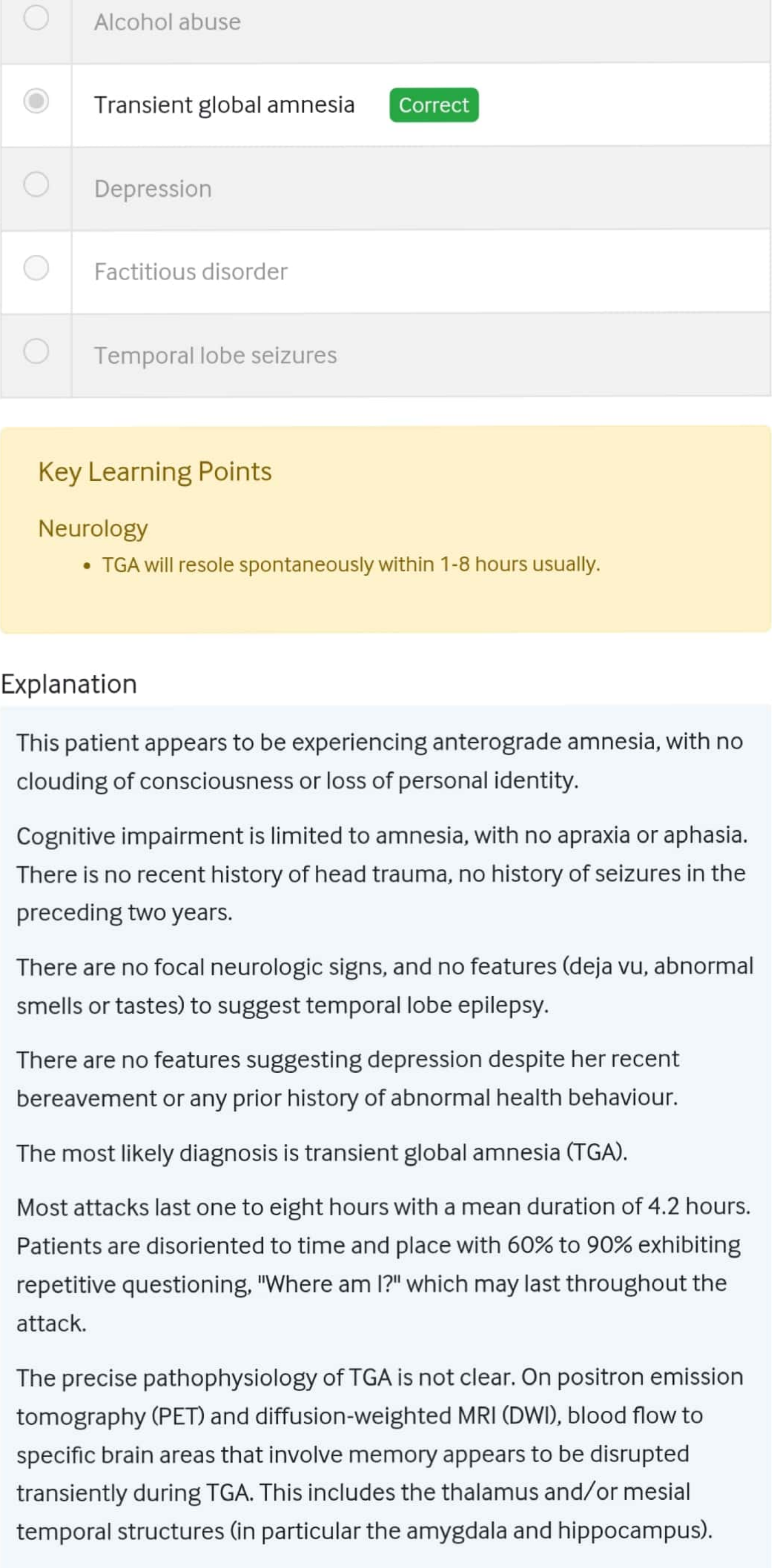


○ Alcohol abuse

● Transient global amnesia **Correct**

○ Depression

○ Factitious disorder

○ Temporal lobe seizures

Key Learning Points

Neurology

- TGA will resole spontaneously within 1-8 hours usually.

Explanation

This patient appears to be experiencing anterograde amnesia, with no clouding of consciousness or loss of personal identity.

Cognitive impairment is limited to amnesia, with no apraxia or aphasia. There is no recent history of head trauma, no history of seizures in the preceding two years.

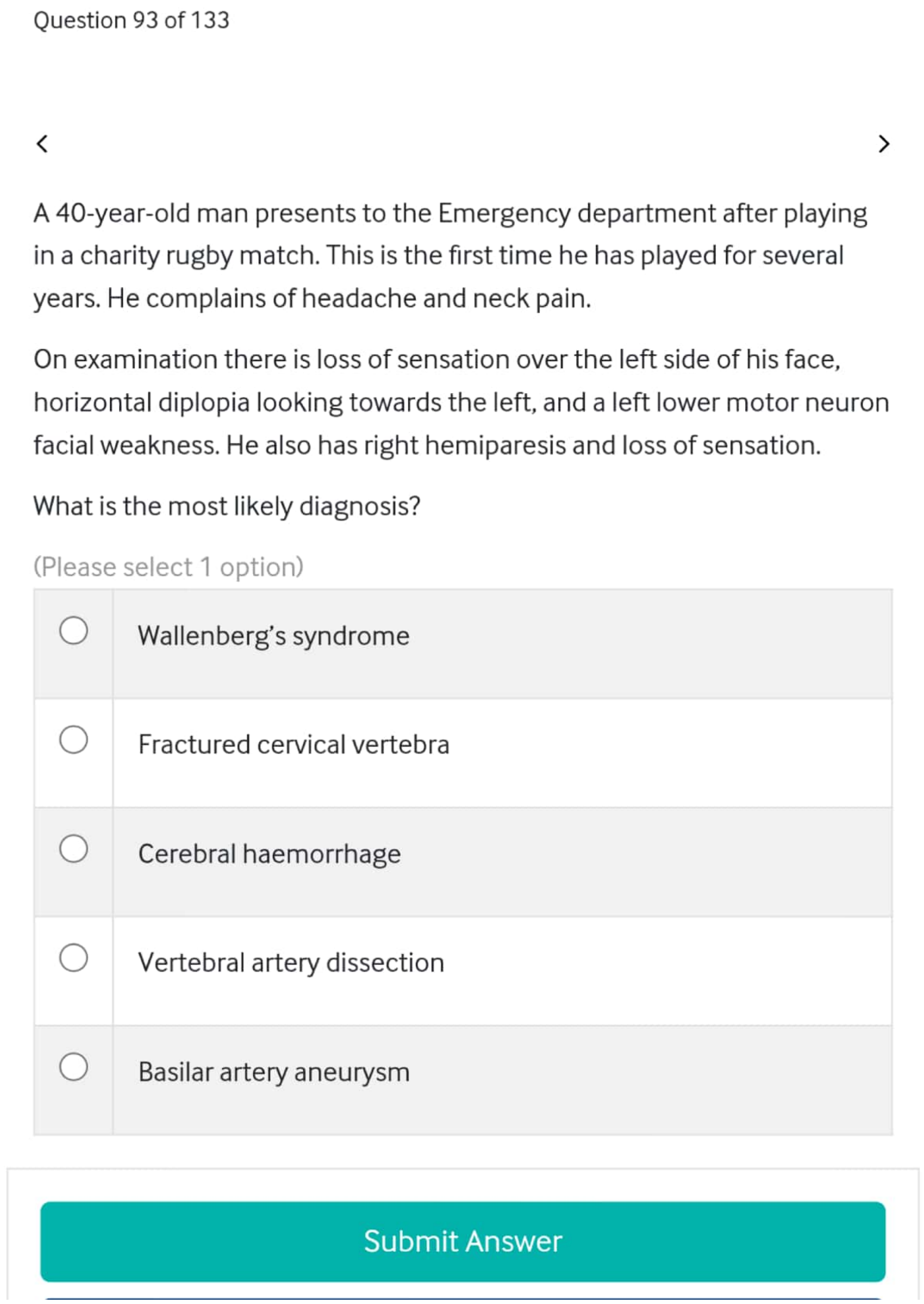
There are no focal neurologic signs, and no features (deja vu, abnormal smells or tastes) to suggest temporal lobe epilepsy.

There are no features suggesting depression despite her recent bereavement or any prior history of abnormal health behaviour.

The most likely diagnosis is transient global amnesia (TGA).

Most attacks last one to eight hours with a mean duration of 4.2 hours. Patients are disoriented to time and place with 60% to 90% exhibiting repetitive questioning, "Where am I?" which may last throughout the attack.

The precise pathophysiology of TGA is not clear. On positron emission tomography (PET) and diffusion-weighted MRI (DWI), blood flow to specific brain areas that involve memory appears to be disrupted transiently during TGA. This includes the thalamus and/or mesial temporal structures (in particular the amygdala and hippocampus).



Question 93 of 133

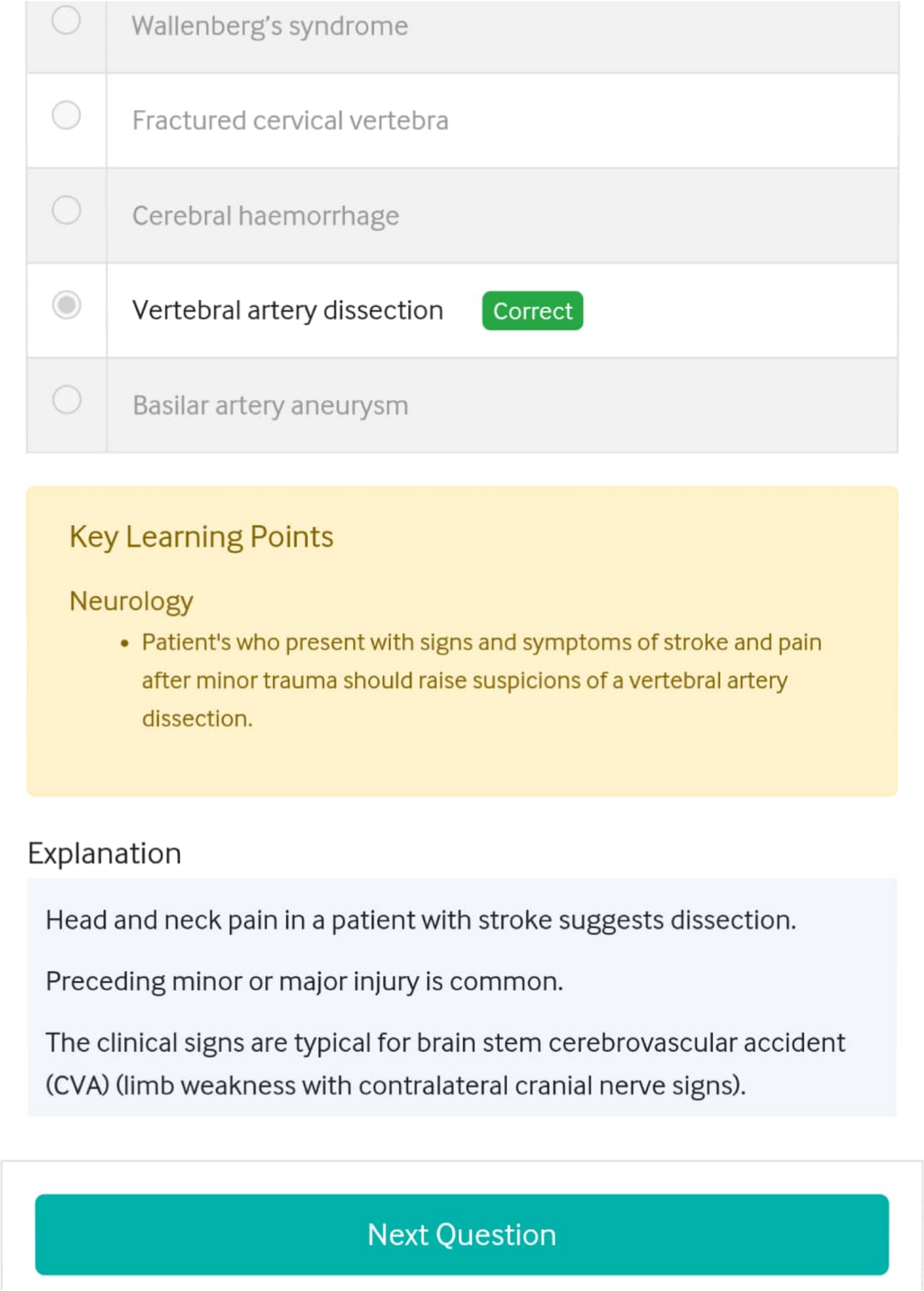
A 40-year-old man presents to the Emergency department after playing in a charity rugby match. This is the first time he has played for several years. He complains of headache and neck pain.

On examination there is loss of sensation over the left side of his face, horizontal diplopia looking towards the left, and a left lower motor neuron facial weakness. He also has right hemiparesis and loss of sensation.

What is the most likely diagnosis?

(Please select 1 option)

○	Wallenberg’s syndrome
○	Fractured cervical vertebra
○	Cerebral haemorrhage
○	Vertebral artery dissection
○	Basilar artery aneurysm



○ Wallenberg’s syndrome

○ Fractured cervical vertebra

○ Cerebral haemorrhage

● Vertebral artery dissection **Correct**

○ Basilar artery aneurysm

Key Learning Points

Neurology

- Patient's who present with signs and symptoms of stroke and pain after minor trauma should raise suspicions of a vertebral artery dissection.

Explanation

Head and neck pain in a patient with stroke suggests dissection.

Preceding minor or major injury is common.

The clinical signs are typical for brain stem cerebrovascular accident (CVA) (limb weakness with contralateral cranial nerve signs).

You are asked to review a 16-year-old Caucasian girl in the Emergency department. She has been unwell for several days, complaining of feeling very tired and having intermittent fevers and chills. Suddenly, 30 minutes before her admission to hospital, she lost all vision in her left eye.

On examination she appears pale and unwell. Vital signs: temperature 38.5°C; pulse 120/minute, regular; blood pressure 100/55 mmHg; respiratory rate 22/minute. A pansystolic murmur is audible at the apex and lower left sternal border. Both lungs are clear.

The right pupil reacts normally to light; there is no reaction from the left pupil which remains fixed and dilated. There is complete loss of vision in the left eye. The left fundus appears paler than the right; there is no papilloedema.

The only additional finding on examination was a paronychia on her right thumb; light pressure on the nail bed was very uncomfortable.

Investigations show:

Hb	109 g/L	(115-165)
WBC	14.1 $\times 10^9$/L	(4-11)
Neutrophils	9.0 $\times 10^9$/L	(1.5-7)
Lymphocytes	4.8 $\times 10^9$/L	(1.5-4)
Monocytes	0.29 $\times 10^9$/L	(0-0.8)
Eosinophils	0.01 $\times 10^9$/L	(0.04-0.4)
Platelets	550 $\times 10^9$/L	(150-400)

What is the most important investigation in determining the cause of her illness?

(Please select 1 option)

○ X ray hand

○ Blood cultures

○ Cerebral angiogram

○ CT head

○ MRI head with gadolinium contrast

Submit Answer

● Blood cultures **Correct**

○ Cerebral angiogram

○ CT head

○ MRI head with gadolinium contrast

Key Learning Points

Neurology

- Early sets of blood cultures remain the most important investigation in a suspected case of infective endocarditis.

Explanation

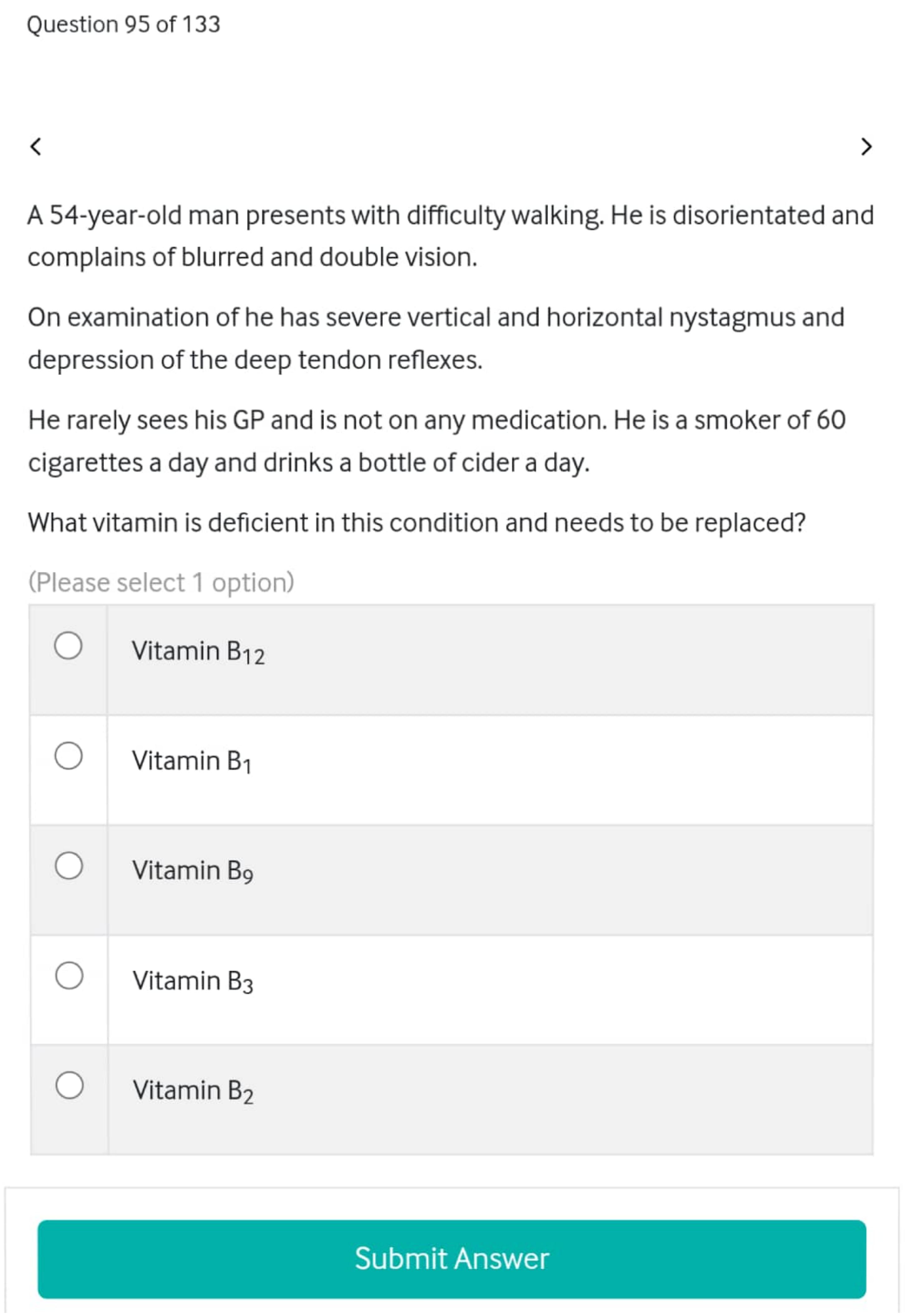
The patient has a chronic paronychia which has caused osteomyelitis of the first phalanx and subsequent endocarditis.

The most likely causative organism is *Staphylococcus aureus*.

Emboli in endocarditis, consisting of vegetation fragments can occlude or damage virtually any blood vessel, large or small, in the systemic or pulmonary arterial circulation. As a result, emboli can produce blindness, stroke or paralysis.

Several of the listed investigations might help in the complete assessment of the patient. However, the essential immediate investigations on which her management depends are blood cultures, followed by echocardiography.

Next Question



A 54-year-old man presents with difficulty walking. He is disorientated and complains of blurred and double vision.

On examination of he has severe vertical and horizontal nystagmus and depression of the deep tendon reflexes.

He rarely sees his GP and is not on any medication. He is a smoker of 60 cigarettes a day and drinks a bottle of cider a day.

What vitamin is deficient in this condition and needs to be replaced?

(Please select 1 option)

○ Vitamin B_{12}

○ Vitamin B_1

○ Vitamin B_9

○ Vitamin B_3

○ Vitamin B_2

○ Vitamin B_{12}

● Vitamin B_1 **Correct**

○ Vitamin B_9

○ Vitamin B_3

○ Vitamin B_2

Key Learning Points

Neurology

- Wernicke-Korsakoff syndrome is as a result of chronic alcohol use and is due to thiamine (vitamin B1) deficiency.

Explanation

This gentleman has ataxia, opthalmoplegia and disorientation associated with Wernicke-Korsakoff syndrome. This is as a result of chronic alcohol use and is due to thiamine (vitamin B_1) deficiency.

Vitamin B_2 or riboflavin deficiency can cause angular cheilitis, glossitis and seborrhoeic dermatitis.

Vitamin B_3 or niacin deficiency can cause dermatitis, dementia and diarrhoea (pellagra).

Vitamin B_9 or folic acid deficiency causes a macrocytic anaemia.

Vitamin B_{12} deficiency causes a macrocytic anaemia, peripheral neuropathy and cognitive impairment.

A 75-year-old man was referred to clinic by his general practitioner following three episodes of transient right monocular visual loss. He described the visual loss as a curtain descending down on the right eye. The visual loss lasted several minutes and resolved. He also reported having recent headaches. He denied having any limb weakness and had been entirely well prior to these events.

He had a past medical history of hypertension and was taking regular bendroflumethiazide. He was a smoker of 30 cigarettes per day and did not drink any alcohol.

On examination he looked plethoric. His blood pressure was 140/80 mmHg, pulse was 88 beats per minute and regular. Oxygen saturation, measured by pulse oxymetry, was 99% on room air.

On examination of the peripheral vascular system, there was an audible bruit heard over the right carotid artery; he had pulsatile non-tender temporal arteries. His heart sounds were normal and his chest was clear.

On examination of his abdomen, a mass was palpated in the left upper quadrant. A full neurological examination, including fundoscopy, was entirely normal.

Investigations revealed:

Haemoglobin	198 g/L	(130 - 180)
White cell count	15 $\times 10^9$/L	(4 - 11)
Platelets	450 $\times 10^9$/L	(150 - 400)
Haematocrit	0.75	(0.40 - 0.52)
Red cell count	8.9 $\times 10^{12}$/L	(3.8 - 5.6)
ESR (Westergren)	<5 mm 1st hour	(0 - 20)
Serum sodium	135 mmol/L	(137 - 144)
Serum potassium	4.3 mmol/L	(3.5 - 4.9)
Serum urea	4.5 mmol/L	(2.5 - 7.5)
Serum creatinine	88 µmol/L	(60 - 110)
Serum cholesterol	6.8 mmol/L	(<5.2)
Plasma glucose	4.5 mmol/L	(3.0 - 6.0)

Doppler scanning of the carotid arteries showed: 100% stenosis of the left internal carotid artery, 60% stenosis of right internal carotid artery. He was commenced on aspirin 300 mg for two weeks orally.

What else would you like to do for this patient?

(Please select 1 option)

- ○ Clopidogrel
- ○ Commence on prednisolone 60 mg
- ○ Venesection
- ○ Right carotid endarterectomy
- ○ Arrange temporal artery biopsy

Submit Answer

● Venesection **Correct**

○ Right carotid endarterectomy

○ Arrange temporal artery biopsy

Key Learning Points

Neurology

- Treatment of stroke secondary to polycythaemia includes venesection.

Explanation

This patient has had several episodes of amaurosis fugax as a result of primary polycythaemia.

In addition he has several risk factors for cerebrovascular disease including hypertension, hypercholesterolaemia and a history of smoking, plethora and splenomegaly and elevated haemoglobin.

Haematocrit and red cell count are all in keeping with the diagnosis of polycythaemia vera.

The two most important treatments in this case would be aspirin and venesection. These two measures should reduce the risk of further transient ischaemic attacks (TIAs) or stroke.

Carotid endarterectomy is considered if there is significant symptomatic carotid artery stenosis, that is between 70% - 99% although some centres will accept >50%.

One hundred percent carotid artery stenosis usually requires a bypass procedure, as risk of endarterectomy outweighs benefit.

Aspirin is the first choice as antiplatelet treatment and given the elevated cholesterol level a statin should be commenced.

A 59-year-old man attended his GP complaining of pain in his left arm.

He noticed the pain particularly when lifting weights in his left hand and during exercising he would become weak and dizzy and nauseated and on two occasions had passed out without warning. He complained of neck pain and had recently had an MR scan of the neck and was told he had some 'wear and tear' changes in some of the vertebrae.

He had a past history of hypertension and hypercholesterolaemia and took regular atenolol and simvastatin. He was a smoker of 20 cigarettes per day and drank approximately 20 units of alcohol per week. He was a builder and was married with two adult children.

On examination he was alert and orientated. His blood pressure was 150/78 mmHg taken in the right arm and 120/67 mmHg in the left arm. His pulse was 80/minute and regular and there was evidence of bilateral carotid bruits. Heart sounds were normal and his chest was clear.

On examination of the neurological system, tone appeared normal, reflexes were depressed at the biceps and brachioradialis with a brisk triceps jerk. There was no demonstrable weakness, however there was some impaired pinprick sensation over the thumb, and middle finger in the left hand. There were no signs of atrophy or fasciculations. Cerebellar system appeared intact.

Carotid Doppler studies revealed:

- 70% stenosis of the left internal carotid artery
- 50% stenosis of the right internal carotid artery.

The chest x ray was normal.

What is the most likely diagnosis in this patient?

(Please select 1 option)

○ Left carotid artery disease

○ Cervical spondylosis

○ Subclavian steal syndrome

○ Pancoast's tumour

○ Thoracic outlet obstruction

Submit Answer

○ Cervical spondylosis

● Subclavian steal syndrome **Correct**

○ Pancoast’s tumour

○ Thoracic outlet obstruction

Key Learning Points

Neurology

- Subclavian steal syndrome occurs when there is an occlusion proximal to the origin of the left vertebral artery and blood is redirected from the right vertebral artery causing basilar insufficiency.

Explanation

This patient presents with a classic history of subclavian steal syndrome brought on by exercising of his left hand, and associated with a reduction in blood pressure in the left arm.

Subclavian steal syndrome occurs when there is an occlusion proximal to the origin of the left vertebral artery. As a result blood is stolen from the right vertebral artery with resultant basilar insufficiency.

This is manifest by brainstem features such as:

- Vertigo
- Diplopia
- Dysarthria, and
- Drop attacks.

Risk factors include:

- Hypertension
- Hypercholesterolaemia
- Diabetes, and
- Connective tissue disorders such as Takayasu's arteritis.

Carotid artery disease would not cause brainstem features or syncope.

There is nothing to suggest a Pancoast's tumour which would cause a thoracic outlet syndrome.

Thoracic outlet syndrome occurs due to damage of the eighth cervical and first thoracic nerve roots (part of brachial plexus) and/or subclavian vessels either by compression over a cervical or abnormal thoracic rib or an aberrant fibrous band. The patient complains of muscle weakness and atrophy of the hand and wrist. There may be pain, loss of sensation and tingling in the medial forearm and little and first fingers often following carrying heavy items.

Occasionally, cyanosis, Raynaud's phenomenon and swelling of arm can occur.

Syncope is not normally part of this syndrome.

Cervical spondylosis (at C5/C6) may explain some of the reflex changes and sensory disturbances in this patient, but would not explain the brainstem features.

Next Question

A 22-year-old woman was seen in the Emergency department with a five day history of progressive weakness and lethargy.

She had initially complained of a constant dry mouth and nausea associated with some swallowing difficulties. However, in the last few days she has noticed blurring of vision and more recently upper limb weakness and breathlessness on exertion. She denied haemoptysis, sputum or cough but did complain of a sore throat.

Preceding her symptoms she had experienced some abdominal pain but thought it may have been due to her period. She denied any medical problems and was a smoker of 20 cigarettes per day.

On examination she was alert and orientated. Her temperature was 36.7°C, blood pressure 110/60 mmHg, pulse 78/min and respiratory rate 20/min. There was evidence of intravenous drug abuse with several track marks and puncture sites found on both thighs. On cranial nerve examination she had mild bilateral ptosis with restricted ocular movements on right lateral and right superior gaze. There were reduced palatal movements and neck extension was weak.

Examination of the upper limb revealed reduced reflexes bilaterally with some proximal weakness, however sensory examination was normal. On examination of the lower limb, reflexes were all depressed, power and sensory examination was normal.

Chest examination was normal other than slightly reduced expansion 4 cm and abdominal examination was entirely normal.

A full blood count and biochemical profile were all normal.

Lumbar puncture:

Opening pressure	12cm H_2O	(5-18)
Protein	0.65 g/L	(0.15-0.45)
White cell count	3 per ml	(≤5)
Red cell count	2 per ml	(≤5)
Tensilon test	Positive	-

What is the likely diagnosis for this patient?

(Please select 1 option)

○ Lambert-Eaton syndrome

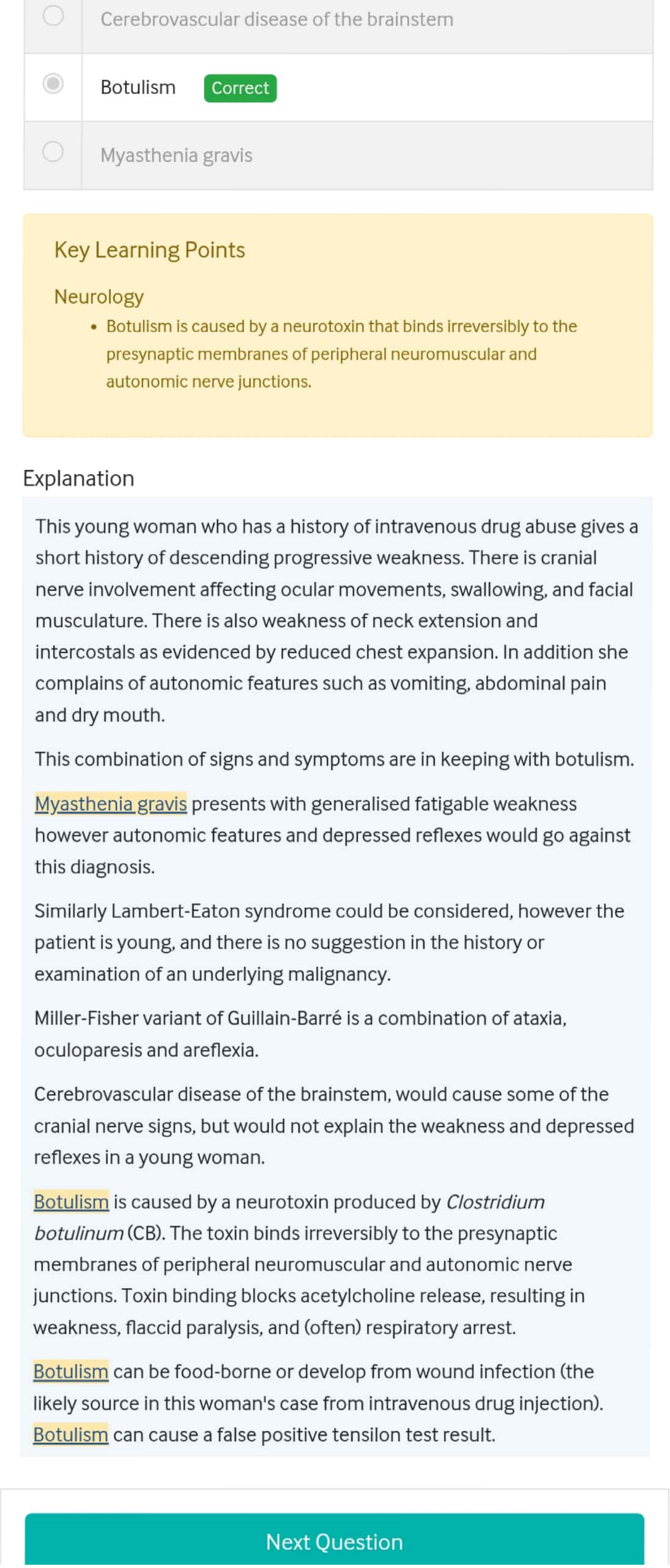
○ Miller-Fisher variant of Guillain-Barré syndrome

○ Cerebrovascular disease of the brainstem

○ Botulism

○ Myasthenia gravis

Submit Answer



○ Cerebrovascular disease of the brainstem

● Botulism **Correct**

○ Myasthenia gravis

Key Learning Points

Neurology

- Botulism is caused by a neurotoxin that binds irreversibly to the presynaptic membranes of peripheral neuromuscular and autonomic nerve junctions.

Explanation

This young woman who has a history of intravenous drug abuse gives a short history of descending progressive weakness. There is cranial nerve involvement affecting ocular movements, swallowing, and facial musculature. There is also weakness of neck extension and intercostals as evidenced by reduced chest expansion. In addition she complains of autonomic features such as vomiting, abdominal pain and dry mouth.

This combination of signs and symptoms are in keeping with botulism.

Myasthenia gravis presents with generalised fatigable weakness however autonomic features and depressed reflexes would go against this diagnosis.

Similarly Lambert-Eaton syndrome could be considered, however the patient is young, and there is no suggestion in the history or examination of an underlying malignancy.

Miller-Fisher variant of Guillain-Barré is a combination of ataxia, oculoparesis and areflexia.

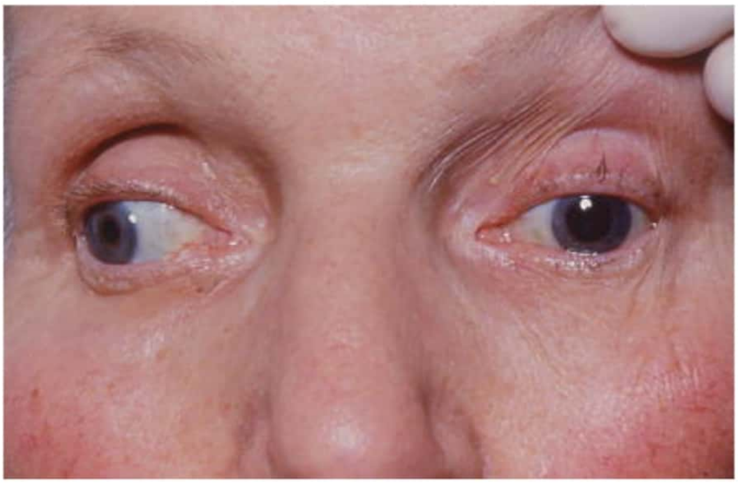
Cerebrovascular disease of the brainstem, would cause some of the cranial nerve signs, but would not explain the weakness and depressed reflexes in a young woman.

Botulism is caused by a neurotoxin produced by *Clostridium botulinum* (CB). The toxin binds irreversibly to the presynaptic membranes of peripheral neuromuscular and autonomic nerve junctions. Toxin binding blocks acetylcholine release, resulting in weakness, flaccid paralysis, and (often) respiratory arrest.

Botulism can be food-borne or develop from wound infection (the likely source in this woman's case from intravenous drug injection). Botulism can cause a false positive tensilon test result.

This patient presents with sudden onset of frontal headache, diplopia and vomiting.

Examination reveals the appearances of the eyes as shown below:



There is also constriction of the temporal visual fields bilaterally, and sensory loss over the upper maxillae bilaterally.

Investigations reveal:

Sodium	128 mmol/L	(137-144)
Potassium	4.2 mmol/L	(3.5-4.9)
Urea	4.2 mmol/L	(2.5-7.5)
Creatinine	67 µmol/L	(60-110)
Plasma glucose	3.8 mmol/L	(3.0-6.0)

Which of the following diagnoses is most likely?

(Please select 1 option)

- ○ Anterior communicating artery aneurysm rupture
- ○ Pituitary apoplexy
- ○ Meningitis
- ○ Lateral medullary syndrome
- ○ Weber's syndrome

Submit Answer

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○ Anterior communicating artery aneurysm rupture

● Pituitary apoplexy Correct

○ Meningitis

○ Lateral medullary syndrome

○ Weber's syndrome

Key Learning Points

Endocrinology, Neurology, Photographic

- Pituitary apoplexy can cause compression of the occular nerves and trigeminal nerve within the cavernous sinus.

Explanation

This patient has a sudden onset headache and features of a cavernous sinus compression (i.e. third and fifth nerve palsy. The patient's left eye has ptosis and hence the clinician is pulling up the eyelid to examine the eye, which shows a dilated pupil. The bilateral numbness over the maxillae indicates involvement of the trigeminal nerve). However compression of the chiasm as suggested by the bitemporal hemianopia. This suggests a diagnosis of pituitary apoplexy.

Apoplexy is a haemorrhage or infarction of the pituitary. The expanding mass can compress the cavernous sinus with the Ocular nerves and the trigeminal nerve can also be affected.

The mild hyponatraemia may be a consequence of the either syndrome of inappropriate antidiuretic hormone (SIADH) or secondary hypoadrenalism.

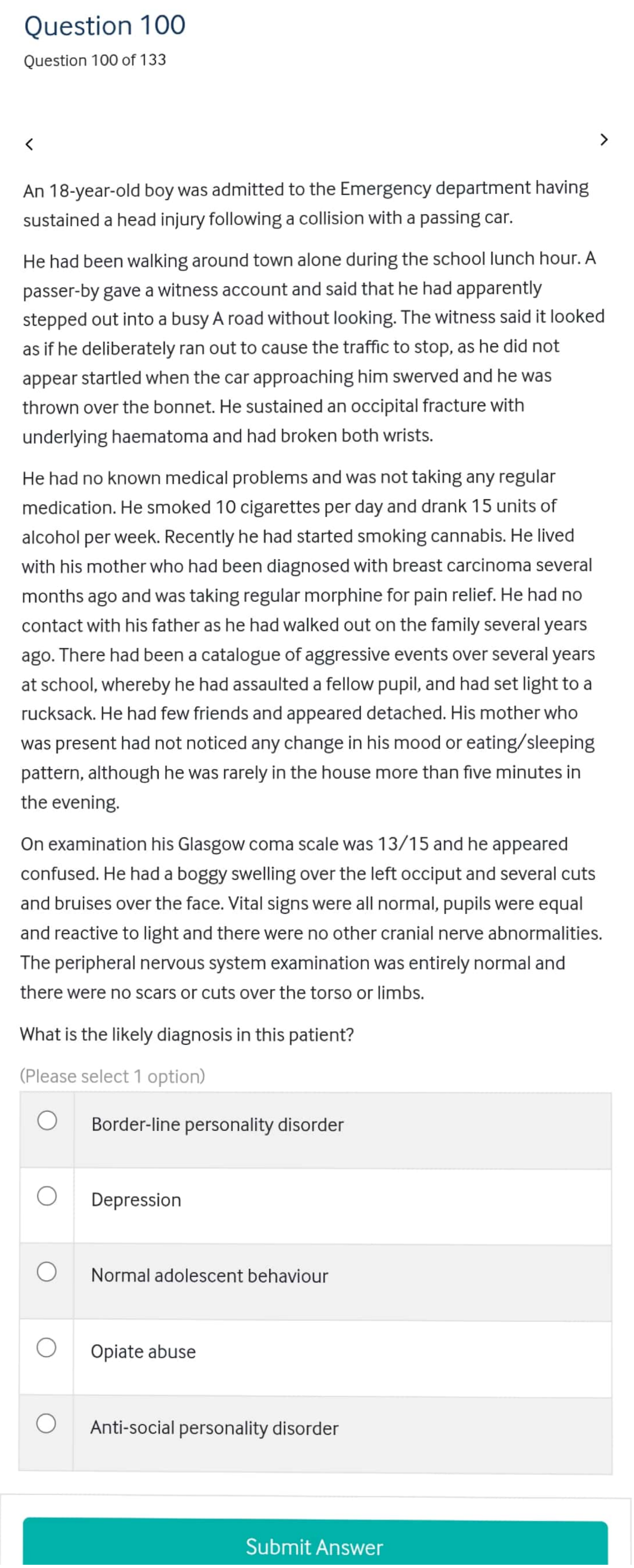
Pituitary apoplexy usually occurs in a pre-existent pituitary tumour which may be entirely asymptomatic before presentation.

Weber's syndrome is a mid brain stroke with ipsilateral third nerve palsy and contralateral hemiplegia.

Most anterior artery communicating aneurysms are asymptomatic unless they rupture, and so they are usually found either incidentally or when a patient presents with SAH. Some unruptured aneurysms can become symptomatic. Symptoms include headache (which may be severe and comparable to the headache of SAH), visual acuity loss, cranial neuropathies (particularly third nerve palsy), pyramidal tract dysfunction, and facial pain; they are felt to be due to the mass effect of the aneurysm. However they would not present with a bitemporal hemianopia as with this patient.

Wallenberg's syndrome, or lateral medullary syndrome, is due to infarction of the posterior inferior cerebellar artery resulting in ipsilateral V, IX, X nerve involvement, dissociated sensory loss and ataxia.

Next Question



Question 100

Question 100 of 133

An 18-year-old boy was admitted to the Emergency department having sustained a head injury following a collision with a passing car.

He had been walking around town alone during the school lunch hour. A passer-by gave a witness account and said that he had apparently stepped out into a busy A road without looking. The witness said it looked as if he deliberately ran out to cause the traffic to stop, as he did not appear startled when the car approaching him swerved and he was thrown over the bonnet. He sustained an occipital fracture with underlying haematoma and had broken both wrists.

He had no known medical problems and was not taking any regular medication. He smoked 10 cigarettes per day and drank 15 units of alcohol per week. Recently he had started smoking cannabis. He lived with his mother who had been diagnosed with breast carcinoma several months ago and was taking regular morphine for pain relief. He had no contact with his father as he had walked out on the family several years ago. There had been a catalogue of aggressive events over several years at school, whereby he had assaulted a fellow pupil, and had set light to a rucksack. He had few friends and appeared detached. His mother who was present had not noticed any change in his mood or eating/sleeping pattern, although he was rarely in the house more than five minutes in the evening.

On examination his Glasgow coma scale was 13/15 and he appeared confused. He had a boggy swelling over the left occiput and several cuts and bruises over the face. Vital signs were all normal, pupils were equal and reactive to light and there were no other cranial nerve abnormalities. The peripheral nervous system examination was entirely normal and there were no scars or cuts over the torso or limbs.

What is the likely diagnosis in this patient?

(Please select 1 option)

○ Border-line personality disorder

○ Depression

○ Normal adolescent behaviour

○ Opiate abuse

○ Anti-social personality disorder

○	Opiate abuse
○	Anti-social personality disorder **This is the correct answer**

Key Learning Points

Neurology

- Anti-social personality disorder presents with a history of violence, impulsive acts and detachment from friends or family.

Explanation

The most likely diagnosis that fits the patient's description is anti-social personality disorder.

It is difficult to know whether there was any suicidal intent in crossing the busy road, as we only have a witness account of the events. In the history however, there are no signs or symptoms suggestive of depression.

He has a history of violence and impulsive acts with associated substance abuse and appears detached with few friends. There are no features in the history or examination to suggest opiate abuse.

Borderline personalities, although impulsive are self damaging normally with reckless spending, variable moods, and have problems with recurrent suicidal threats or behaviour.

This patient does not depict normal adolescent behaviour.

Next Question

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A 35-year-old rugby player presented to the Emergency department with acute onset pain at the base of the neck and shooting pain affecting the lateral aspect of his left arm to the elbow.

Since the onset of pain, he had also developed some proximal weakness of the left arm and diminished sensation over the deltoid. He had no past medical history and did not take any regular medication. He drank 21 units of alcohol per week and smoked 10 cigarettes per day.

On examination he had some nuchal rigidity on testing neck movements. Lateral flexion to the left appeared to bring on the left sided shooting pain. On examination of the upper limb, tone appeared normal, however, there was reduced power of 4-/5 of elbow flexion and some weakness of shoulder abduction. The left bicep and supinator reflexes were depressed but the triceps reflex appeared intact. There was a patch of sensory loss over the upper lateral aspect of the left arm. Lower limb examination was entirely normal. A plain x ray of the cervical spine was normal.

What is the most likely explanation of his symptoms?

(Please select 1 option)

○ C5 radiculopathy secondary to lateral disc protrusion

○ Central disc protrusion

○ Cervical spondylotic radiculo-myelopathy

○ Neuralgic amyotrophy

○ C6 radiculopathy

● C5 radiculopathy secondary to lateral disc protrusion
Correct

○ Central disc protrusion

○ Cervical spondylotic radiculo-myelopathy

○ Neuralgic amyotrophy

○ C6 radiculopathy

Key Learning Points

Neurology

- Isolated sensory dermatomal distribution is usually suggestive of a cervical disc prolapse at a certain level.

Explanation

This patient presents with neck pain and shooting pain in a distribution typical for C5 root disturbance.

In addition, there is significant compression leading to weakness of biceps and deltoid muscles and depression of the biceps and supinator reflexes, which are supplied by C5/C6. The distribution of sensory loss would also fit with a C5 radiculopathy.

The lower limb appears intact, which would go against a diagnosis of central disc protrusion leading to cord compression and spastic leg weakness.

Cervical spondylotic radiculo-myelopathy is unlikely given the age of the patient.

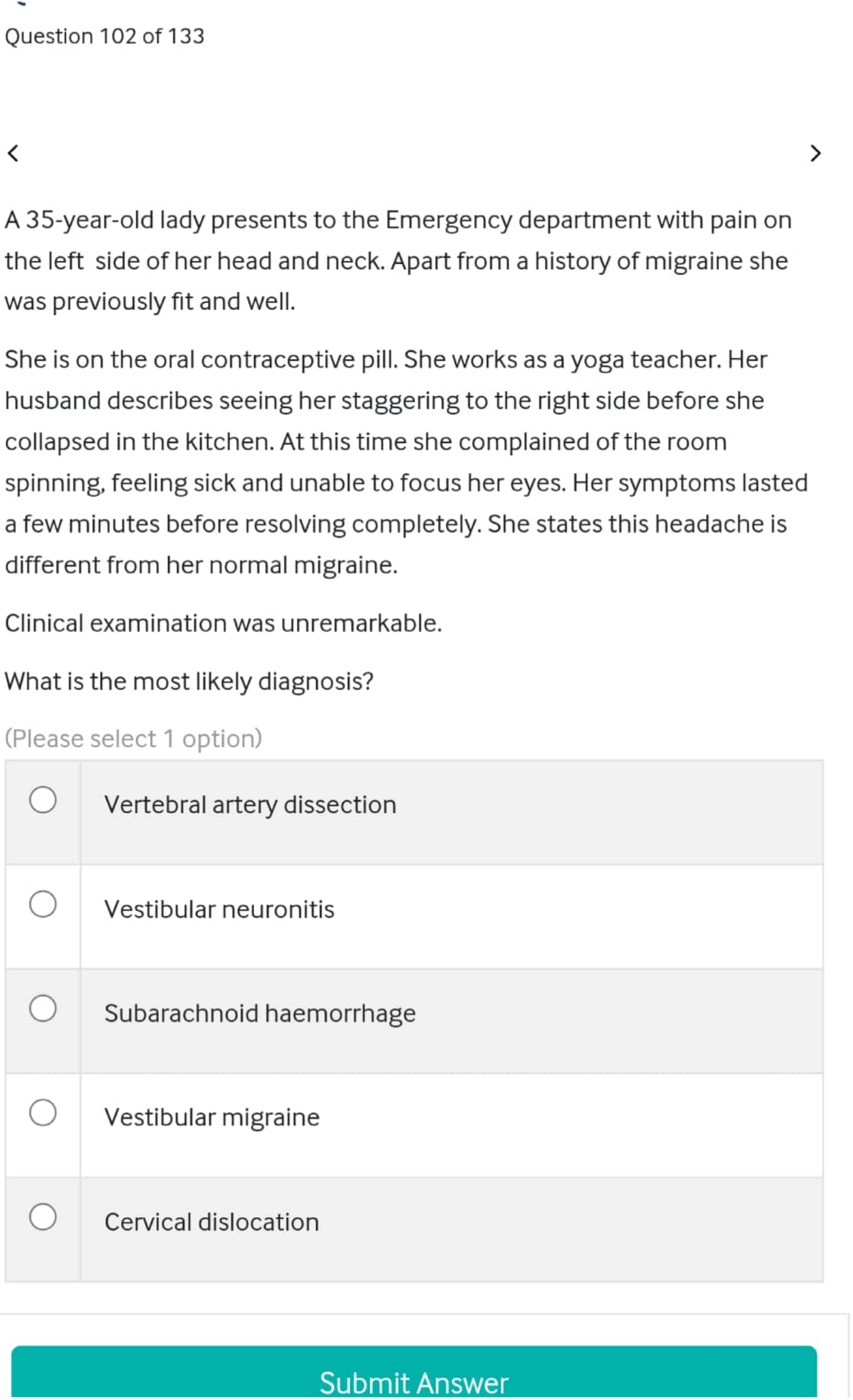
Localising features for a C6 radiculopathy include

- Pain radiating down the lateral arm to the thumb and index finger
- Sensory disturbance affecting the lateral arm, forearm, thumb and index finger and
- Reduced power of biceps and brachioradialis.

The supinator and biceps reflexes would similarly be reduced as in a C5 radiculopathy.

Neuralgic amyotrophy is an acute inflammatory disturbance of the upper brachial plexus (C5/C6) thought secondary to a viral infection.

Next Question



Question 102 of 133

A 35-year-old lady presents to the Emergency department with pain on the left side of her head and neck. Apart from a history of migraine she was previously fit and well.

She is on the oral contraceptive pill. She works as a yoga teacher. Her husband describes seeing her staggering to the right side before she collapsed in the kitchen. At this time she complained of the room spinning, feeling sick and unable to focus her eyes. Her symptoms lasted a few minutes before resolving completely. She states this headache is different from her normal migraine.

Clinical examination was unremarkable.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Vertebral artery dissection
- ○ Vestibular neuronitis
- ○ Subarachnoid haemorrhage
- ○ Vestibular migraine
- ○ Cervical dislocation

(Please select 1 option)

- ● Vertebral artery dissection **Correct**
- ○ Vestibular neuronitis
- ○ Subarachnoid haemorrhage
- ○ Vestibular migraine
- ○ Cervical dislocation

Key Learning Points

Neurology

- Yoga or other stretching exercises where injury may be trivial and not recalled the patient can be a risk factor for developing vertebral artery dissection.

Explanation

Vertebral artery dissection presents with symptoms and signs of brainstem stroke or transient ischaemic attack with associated pain in the ipsilateral neck, face or head. Commonly occurring in young people following a recent head or neck injury, sometimes this injury may be trivial and not recalled by the patient. Yoga can be a risk factor for developing vertebral artery dissection.

Cervical dislocation can occur following trauma but causes injury to the spinal cord and would not present with brainstem symptoms.

Subarachnoid haemorrhage similarly does not present with posterior circulation symptoms and signs but classically with a thunderclap headache and neck stiffness.

Vestibular migraine occurs in individuals who suffer from migraines and is characterised by episodes of vertigo.

Vestibular neuronitis commonly occurs following a viral illness with sudden onset vertigo, nausea and vomiting.

Both are possible diagnoses but in the presence of possible trauma from yoga and in the presence of head and neck pain, they are less likely than vertebral artery dissection.

Next Question

< >

A 30-year-old man presents to the Emergency department with a history of epilepsy.

He starts to experience jerking movements of the angle left of his mouth and then progresses to having jerking movements of his left thumb and index finger. Finally, his entire left side develops jerking movements which subside in ten minutes.

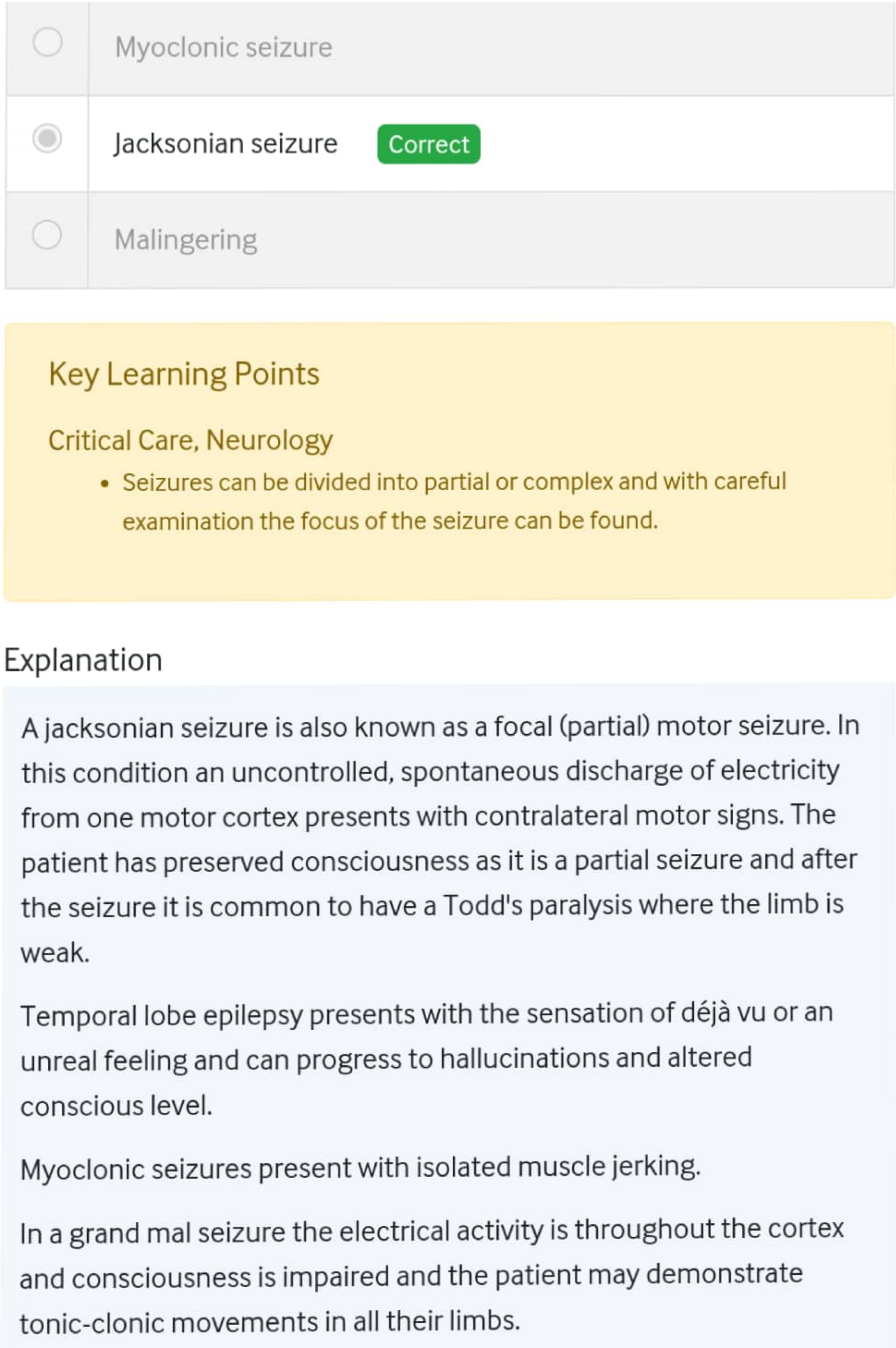
After this he has a weakness down the left side for several hours. During the event he had full consciousness.

What is the likely diagnosis?

(Please select 1 option)

○	Temporal lobe seizure
○	Grand mal seizure
○	Myoclonic seizure
○	Jacksonian seizure
○	Malingering

Submit Answer



○ Myoclonic seizure

● Jacksonian seizure **Correct**

○ Malingering

Key Learning Points

Critical Care, Neurology

- Seizures can be divided into partial or complex and with careful examination the focus of the seizure can be found.

Explanation

A jacksonian seizure is also known as a focal (partial) motor seizure. In this condition an uncontrolled, spontaneous discharge of electricity from one motor cortex presents with contralateral motor signs. The patient has preserved consciousness as it is a partial seizure and after the seizure it is common to have a Todd's paralysis where the limb is weak.

Temporal lobe epilepsy presents with the sensation of déjà vu or an unreal feeling and can progress to hallucinations and altered conscious level.

Myoclonic seizures present with isolated muscle jerking.

In a grand mal seizure the electrical activity is throughout the cortex and consciousness is impaired and the patient may demonstrate tonic-clonic movements in all their limbs.

A 70-year-old male presented to his GP with an odd crawling sensation in his legs and a desire to move them particularly at night.

The symptoms had been present for six months. He found that rubbing his legs or walking around gave him some relief, but the problem had deteriorated over the last two months to the point where he was experiencing daytime somnolence.

He had a past history of hypertension for which he took atenolol and ramipril. He had stopped smoking 10 years ago and drank 20 units of alcohol per week. He lived alone and had little family.

On examination, his blood pressure was 158/88 mmHg, pulse was 80/min and regular and heart sounds were normal. There were no abnormalities of the cranial nerves. On examination of the lower limbs there were several scratch marks. Tone, power and reflexes all appeared normal with flexor plantar responses. All pulses were present and easily palpable. Abdominal examination was entirely normal.

Investigations showed:

Haemoglobin	101 g/L	(130-180)
Mean cell volume	72 fL	(80-96)
White cell count	9.2 $\times 10^9$/L	(4-11)
Platelets	285 $\times 10^9$/L	(150-400)
Serum sodium	138 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	5.1 mmol/L	(2.5-7.5)
Serum creatinine	90 μmol/L	(60-110)
Fasting plasma glucose	4.5 mmol/L	(3-6)
Serum cholesterol	5.1 mmol/L	(<5.2)

What would be your management for this patient?

(Please select 1 option)

○ Commence on aspirin

○ Commence on ferrous sulphate tablets

○ Stop the atenolol

○ Commence on Sinemet

○ Start on amitryptiline

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○ Commence on aspirin

● Commence on ferrous sulphate tablets Correct

○ Stop the atenolol

○ Commence on Sinemet

○ Start on amitryptiline

Key Learning Points

Neurology

- Restless leg syndrome is associated with Fe deficiency, folate def, magnesium def, parkinsons diesase, venous insufficiency and COPD.

Explanation

This patient has restless leg syndrome secondary to iron deficiency anaemia.

This is a condition characterised by uncomfortable, irritating and sometimes painful sensations, which usually occur in the legs when at rest and usually at night. The condition is associated with nutritional deficiencies of folate, magnesium, iron and other conditions such as Parkinson's disease, venous insufficiency and chronic obstructive pulmonary disease (COPD).

It can be exacerbated by diuretics, tricyclics, phenytoin and calcium antagonists.

Differential diagnoses include nocturnal leg cramps, peripheral neuropathy and vascular disease. Nocturnal leg cramps are typically unilateral, painful, palpable, involuntary muscle contractions that are often local with a sudden onset.

In this case, ferrous sulphate therapy should be considered first and further investigation into iron deficiency anaemia should be carried out.

Dopamine agonists and L-dopa should be considered second line symptomatic agents unless the diagnosis is primary restless leg syndrome.

Next Question

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A 20-year-old woman was referred to the outpatient clinic with a two month history of mild exertional dyspnoea.

She reported that her symptoms were more prominent when she took her dog for a walk in the evening; she did not feel breathless when she walked the dog in the morning. Her husband had noted that her speech was sometimes slurred when she returned home after the evening walk and that she looked depressed. She had a past history of mild anxiety and depression that had been treated by her general practitioner.

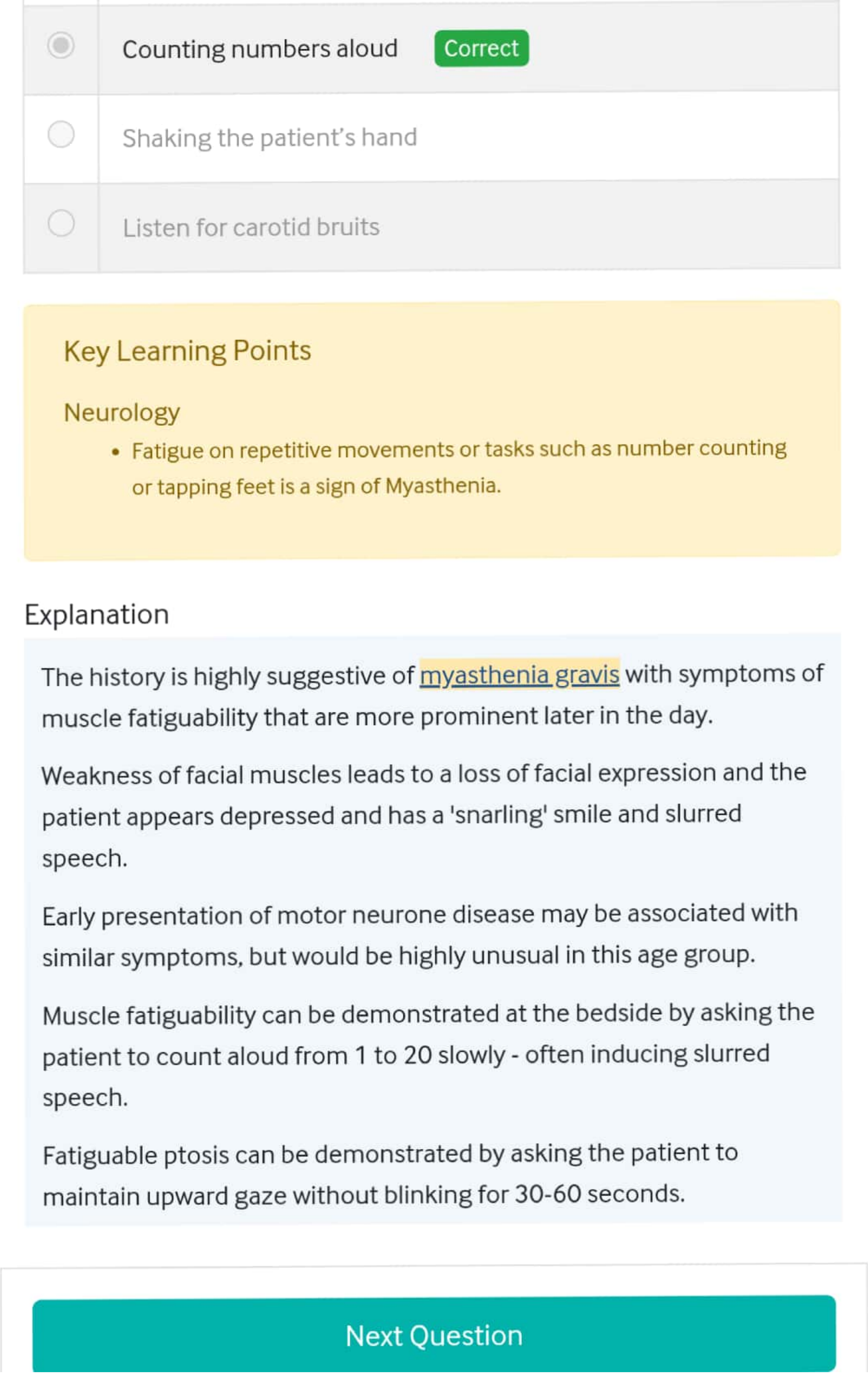
On examination she appeared anxious. There was no palpable lymphadenopathy. Her heart sounds were normal and her chest was clear on auscultation. Her abdomen was soft and non-tender with no palpable masses or organs. Cranial nerves were intact. Tone, bulk and power were normal in all four limbs and her reflexes were brisk and symmetrical with bilateral flexor plantar responses.

What bedside test might be employed to aid in confirming the diagnosis?

(Please select 1 option)

- ○ Inspecting the tongue for fasciculation
- ○ Palpating the neck while asking the patient to swallow
- ○ Counting numbers aloud
- ○ Shaking the patient’s hand
- ○ Listen for carotid bruits

Submit Answer



● Counting numbers aloud **Correct**

○ Shaking the patient's hand

○ Listen for carotid bruits

Key Learning Points

Neurology

- Fatigue on repetitive movements or tasks such as number counting or tapping feet is a sign of Myasthenia.

Explanation

The history is highly suggestive of myasthenia gravis with symptoms of muscle fatiguability that are more prominent later in the day.

Weakness of facial muscles leads to a loss of facial expression and the patient appears depressed and has a 'snarling' smile and slurred speech.

Early presentation of motor neurone disease may be associated with similar symptoms, but would be highly unusual in this age group.

Muscle fatiguability can be demonstrated at the bedside by asking the patient to count aloud from 1 to 20 slowly - often inducing slurred speech.

Fatiguable ptosis can be demonstrated by asking the patient to maintain upward gaze without blinking for 30-60 seconds.

A 35-year-old man was admitted to the Emergency department having been assaulted in the city centre. He had sustained a number of knife wounds, two of which were in the left groin, the rest were superficial cuts. On admission his vital signs were stable and there was bleeding and bruising around the left groin area.

He was a previously fit young man and was not taking any regular medication. He smoked 10 cigarettes per day and drank approximately 20 units of alcohol per week.

On examination, cranial nerves and upper limb examination were entirely normal. Examination of the lower limb revealed good femoral pulses and other distal pulses. Tone appeared normal in both legs. There was reduced power of hip flexion and knee extension on the left, however hip extension and hip adduction and abduction were entirely normal. Power was normal in the right leg. There was a reduced knee jerk on the left, however the rest of the reflexes and plantar responses were normal. Examination of sensation revealed reduced light touch and pinprick over the medial aspect of the left thigh; otherwise there were no other abnormalities.

Investigations revealed:

Full blood count	Normal	-
Biochemistry	Normal	-
Glucose	5.6 mmol/L	(3.0-6.0)
Prothrombin time	11.5 s	(11.5-15.5)

A plain x ray of the pelvis was normal, with no evidence of fracture.

What is the likely cause for this patients symptoms?

(Please select 1 option)

- ○ Sciatic neuropathy
- ○ Lumbar plexopathy
- ○ Femoral neuropathy
- ○ Obturator neuropathy
- ○ L1 root lesion

Submit Answer

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○	Femoral neuropathy **This is the correct answer**
○	Obturator neuropathy
○	L1 root lesion

Key Learning Points

Neurology

- Femoral neuropathy causes weakness predominantly of the quadriceps.

Explanation

Femoral neuropathy causes weakness predominantly of the quadriceps.

The femoral nerve is formed by L2-4 roots as part of the lumbar plexus and is predisposed to compression within the psoas muscle. This occurs as a result of haemorrhage into this muscle due to haemophilia, anticoagulation therapy, or trauma.

The main motor component innervates the iliopsoas (a hip flexor) and the quadriceps (a knee extensor). The motor branch to the iliopsoas originates in the pelvis proximal to the inguinal ligament.

The sensory branch of the femoral nerve, the saphenous nerve, innervates skin of the medial thigh and the anterior and medial aspects of the calf.

Diabetic patients have an unusual predilection for femoral and proximal mononeuropathies.

The obturator nerve innervates the adductors of the hip which are intact in this patient. The sciatic nerve innervates the hip extensors and all muscle compartments below the knee which are all intact in this patient.

A lumbar plexopathy could potentially cause similar physical signs but deep seated excruciating pain is common.

An L1 root lesion would not explain weakness of hip flexion and knee extension.

Next Question

A 45-year-old man presented with diplopia, dysarthria and difficulty with swallowing.

Over the next few days he developed weakness of the upper and lower limbs. On day four he was unable to walk unaided. He denied any sensory symptoms or bladder disturbances. His previous medical history is unremarkable. He is a non-smoker, does not drink alcohol excessively. He does not take any drugs.

On examination he was apyrexial. His general medical examination was normal. His higher mental function was unremarkable. There were no signs of meningism. Cranial nerve examination showed bilateral dilated and fixed pupils. He had binocular diplopia but no obvious ophthalmoplegia. He was dysarthric with weak cough. His vital capacity was 3.15 standing and 2.00 lying flat. He had lower motor neurone tetraparesis of power 3/5. He was hyporeflexic with normal sensation and was unable to walk unaided.

Investigations including full blood count, urea and electrolytes, liver function tests, serum calcium, thyroid function tests, autoantibody screen, erythrocyte sedimentation rate and serum C reactive protein were normal.

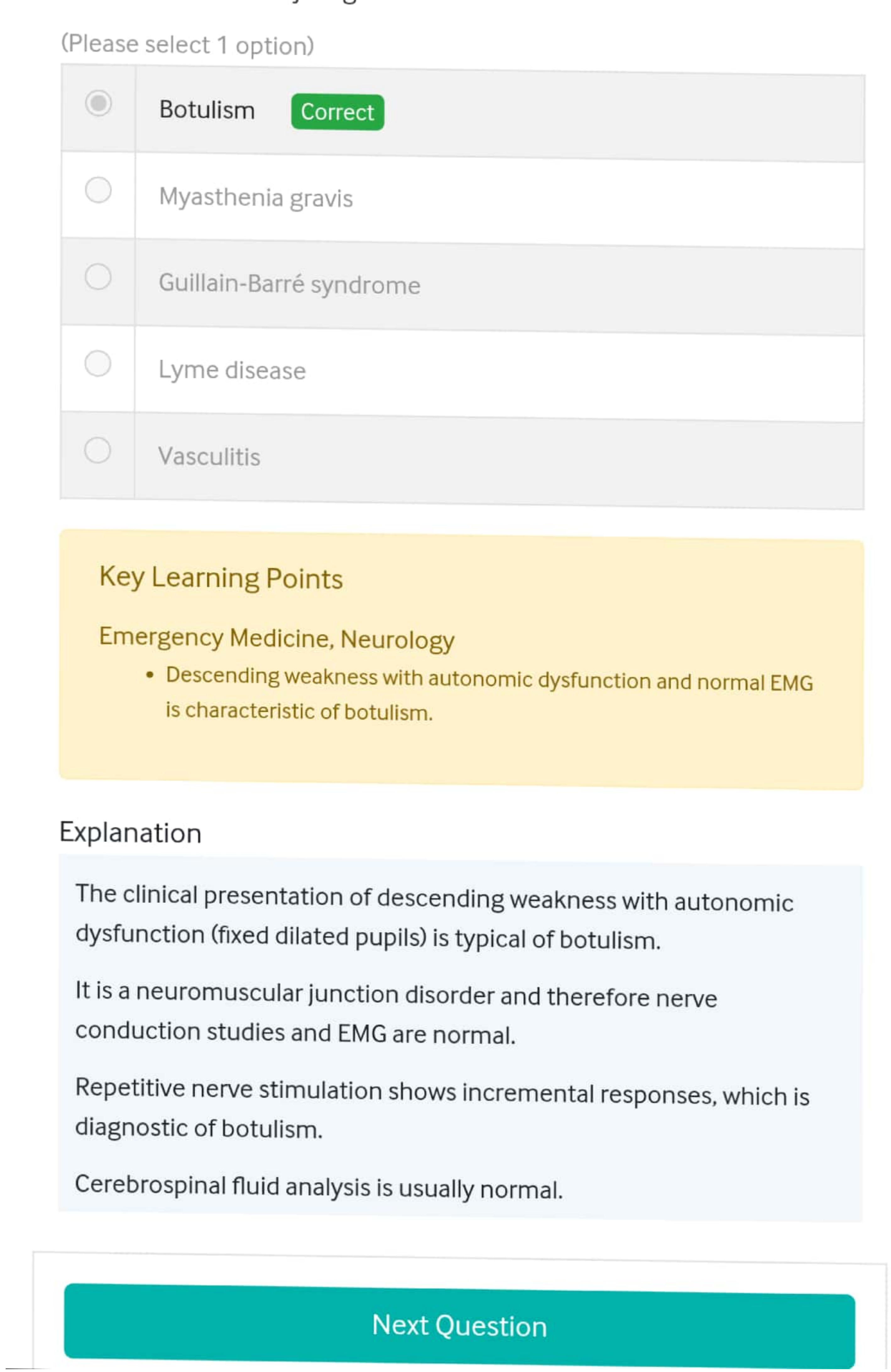
His electrocardiogram and chest radiograph were normal. A CT scan of his brain was normal, as were nerve conduction studies and an electromyogram (EMG).

What is the most likely diagnosis?

(Please select 1 option)

○	Botulism
○	Myasthenia gravis
○	Guillain-Barré syndrome
○	Lyme disease
○	Vasculitis

Submit Answer



(Please select 1 option)

● Botulism **Correct**

○ Myasthenia gravis

○ Guillain-Barré syndrome

○ Lyme disease

○ Vasculitis

Key Learning Points

Emergency Medicine, Neurology

- Descending weakness with autonomic dysfunction and normal EMG is characteristic of botulism.

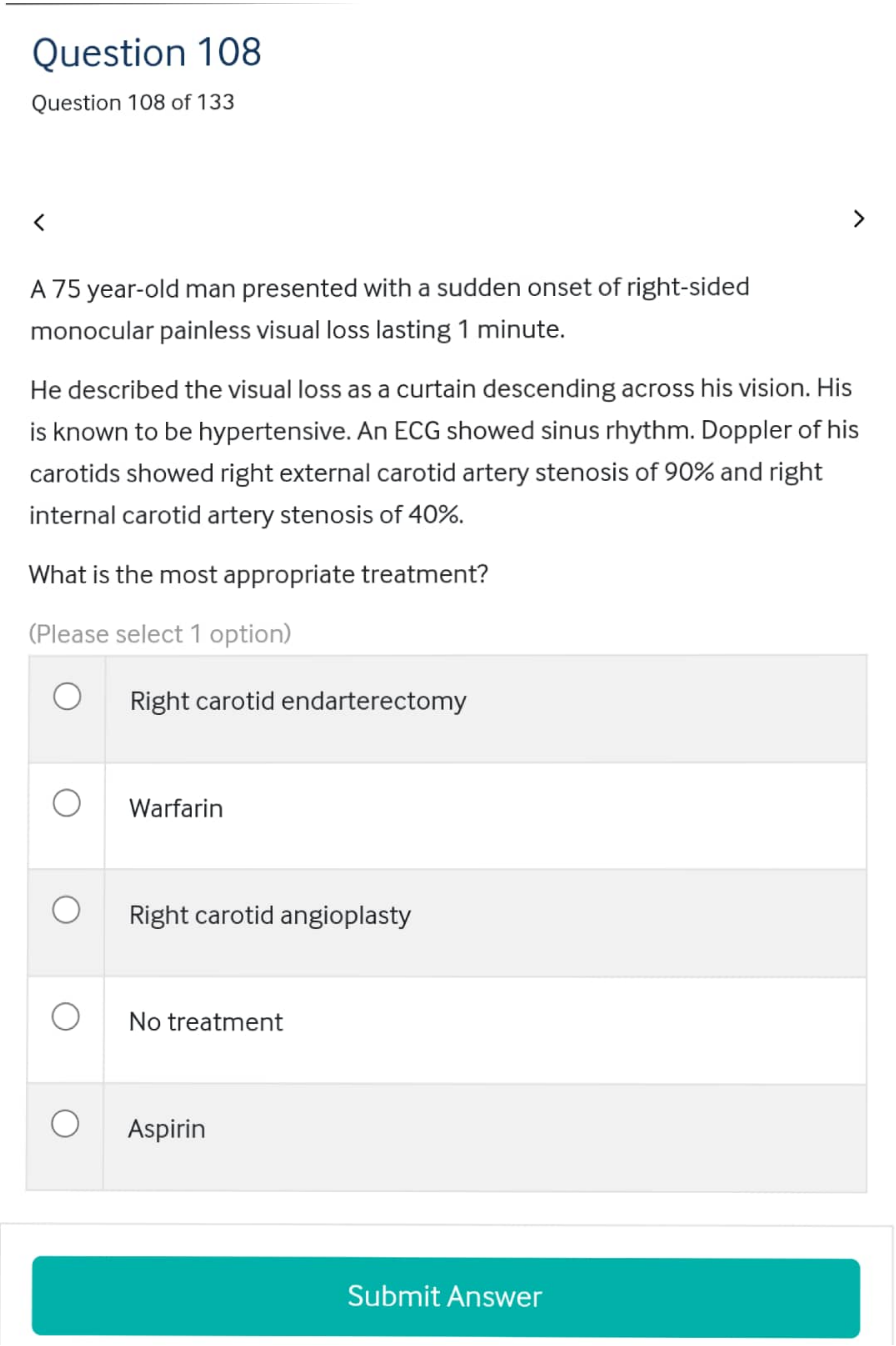
Explanation

The clinical presentation of descending weakness with autonomic dysfunction (fixed dilated pupils) is typical of botulism.

It is a neuromuscular junction disorder and therefore nerve conduction studies and EMG are normal.

Repetitive nerve stimulation shows incremental responses, which is diagnostic of botulism.

Cerebrospinal fluid analysis is usually normal.



Question 108

Question 108 of 133

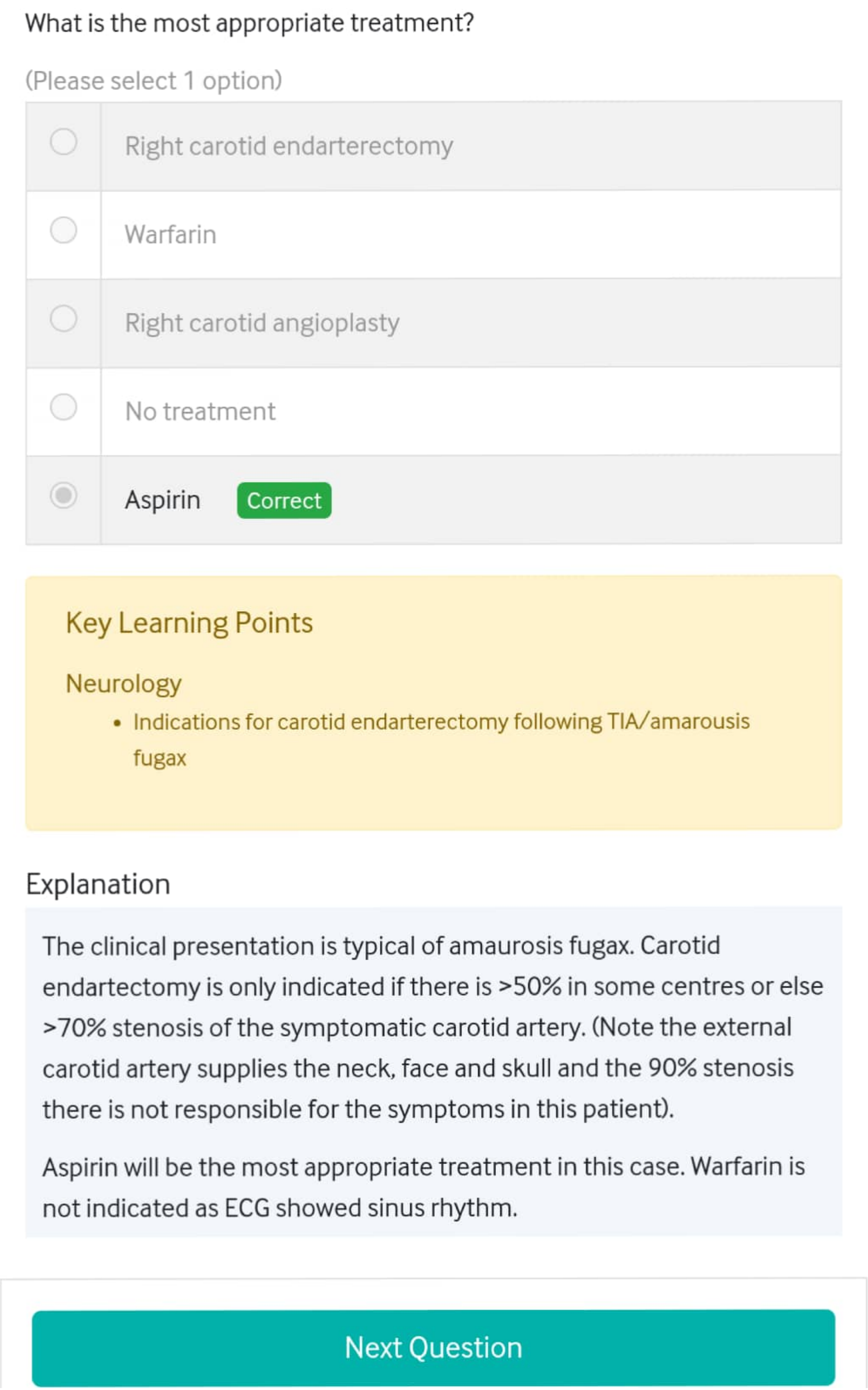
A 75 year-old man presented with a sudden onset of right-sided monocular painless visual loss lasting 1 minute.

He described the visual loss as a curtain descending across his vision. His is known to be hypertensive. An ECG showed sinus rhythm. Doppler of his carotids showed right external carotid artery stenosis of 90% and right internal carotid artery stenosis of 40%.

What is the most appropriate treatment?

(Please select 1 option)

○	Right carotid endarterectomy
○	Warfarin
○	Right carotid angioplasty
○	No treatment
○	Aspirin



What is the most appropriate treatment?

(Please select 1 option)

○	Right carotid endarterectomy
○	Warfarin
○	Right carotid angioplasty
○	No treatment
●	Aspirin Correct

Key Learning Points

Neurology

- Indications for carotid endarterectomy following TIA/amarousis fugax

Explanation

The clinical presentation is typical of amaurosis fugax. Carotid endartectomy is only indicated if there is >50% in some centres or else >70% stenosis of the symptomatic carotid artery. (Note the external carotid artery supplies the neck, face and skull and the 90% stenosis there is not responsible for the symptoms in this patient).

Aspirin will be the most appropriate treatment in this case. Warfarin is not indicated as ECG showed sinus rhythm.

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A 30-year-old man presented to his general practitioner with a one week history of recurrent episodes of severe pain behind the right eye. The pain appeared to come on at night and lasted approximately 90 minutes.

During the preceding week he had had approximately eight attacks. He had not noticed any visual disturbances or diplopia but during an attack his right eye would be watery and red. He had vomited several times with the pain.

He had no known past medical history and did not take any regular medications. He was a smoker of 25 cigarettes per day and drank 30 units of alcohol per week.

On examination he appeared flushed and to be in some distress. His blood pressure was 145/90 mmHg, pulse was 97/min and regular and temperature was 36.7°C. There was a right ptosis and miosis and there was some right conjunctival injection. He had pulsatile non-tender arteries. Pupillary responses appeared intact and ocular movements were full. The rest of the cranial nerve examination was normal. There were no abnormalities detected in the peripheral nervous system or cardiovascular systems.

Investigations revealed:

Haemoglobin	145 g/L	(130-180)
White cell count	11 $\times 10^9$/L	(4-11)
Platelets	345 $\times 10^9$/L	(150-400)
ESR (Westergren)	5 mm/1st hour	(0-15)
Cholesterol	5.8 mmol/L	(< 5.2)
Glucose	4.5 mmol/L	(3.0-6.0)

Which of the following is the best treatment to give during an acute episode?

(Please select 1 option)

○ Prednisolone 60 mg daily for five days

○ Ergotamine

○ Indomethacin

○ Amitriptyline

○ Sumatriptan

Submit Answer

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● Sumatriptan **Correct**

Key Learning Points

Neurology

- NICE guidance suggest managing cluster headache with subcutaneous or intranasal sumatriptan as first line.

Explanation

This patient presents with episodes of nocturnal retro-orbital pain associated with autonomic features (lacrimation, Horner's syndrome, and facial flushing).

These features would be in keeping with a diagnosis of cluster headache. It usually presents in male patients with a male:female ratio of 9:1 and a mean age of onset of 30 years. The attacks are often nocturnal and are associated with parasympathetic overactivity.

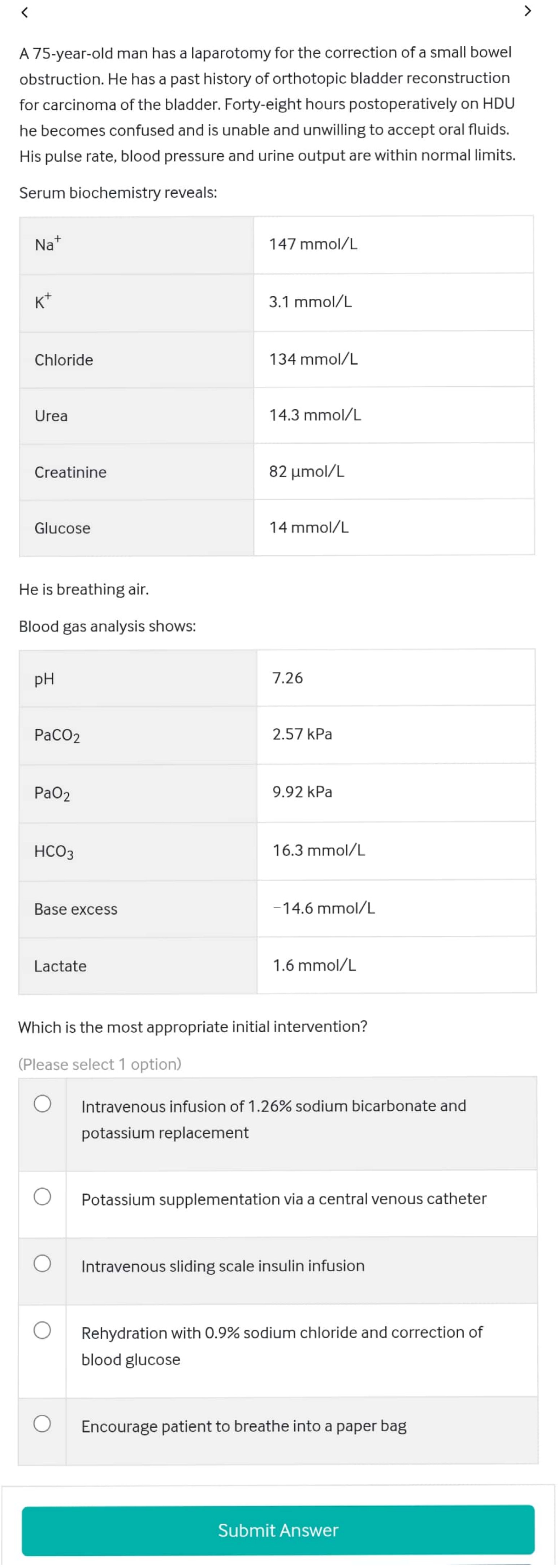
The treatment of choice during the acute attack includes subcutaneous or intranasal sumatriptan and high flow oxygen.

Prophylactic treatment includes:

- Lithium
- Verapamil
- Sodium valproate
- Prednisolone, and
- Ergotamines.

There is nothing in the history or clinical findings to suggest temporal arteritis or optic neuritis warranting further investigations.

Next Question



A 75-year-old man has a laparotomy for the correction of a small bowel obstruction. He has a past history of orthotopic bladder reconstruction for carcinoma of the bladder. Forty-eight hours postoperatively on HDU he becomes confused and is unable and unwilling to accept oral fluids. His pulse rate, blood pressure and urine output are within normal limits.

Serum biochemistry reveals:

Na^+	147 mmol/L
K^+	3.1 mmol/L
Chloride	134 mmol/L
Urea	14.3 mmol/L
Creatinine	82 µmol/L
Glucose	14 mmol/L

He is breathing air.

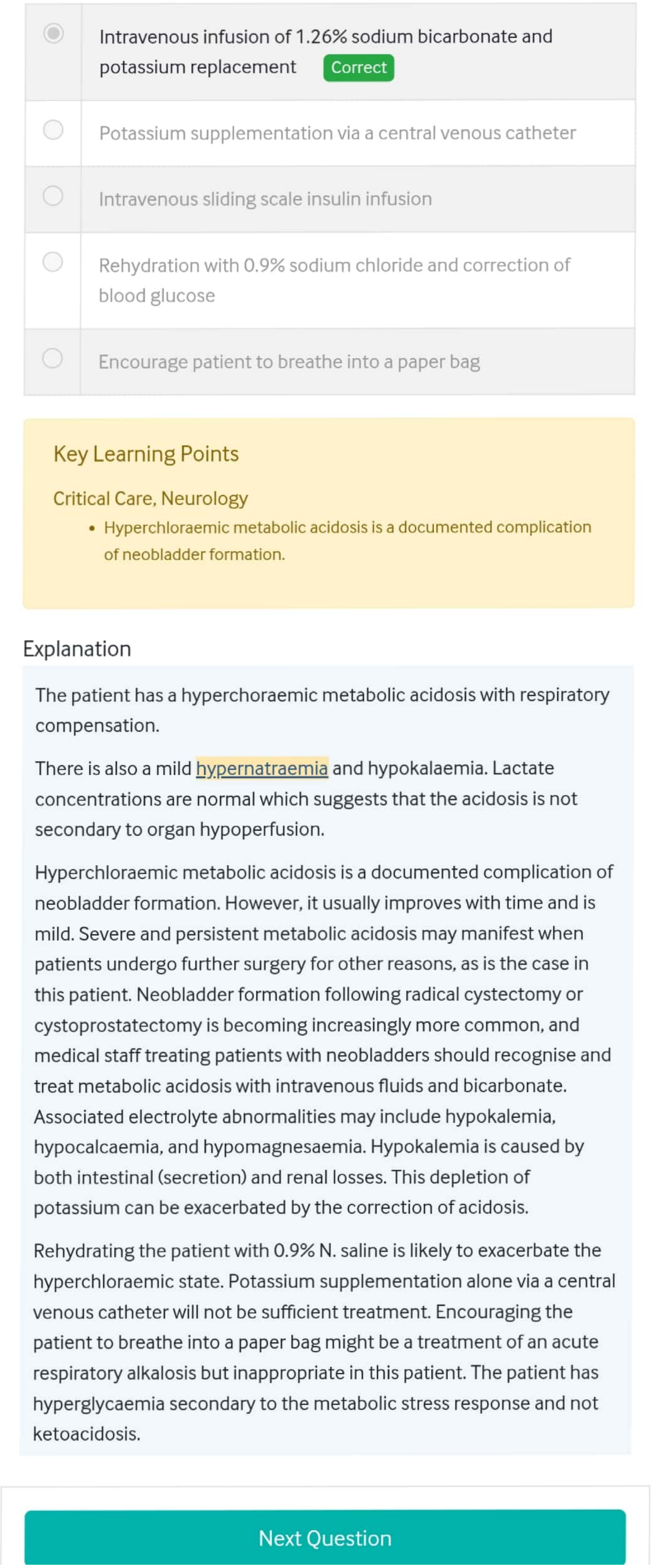
Blood gas analysis shows:

pH	7.26
$PaCO_2$	2.57 kPa
PaO_2	9.92 kPa
HCO_3	16.3 mmol/L
Base excess	−14.6 mmol/L
Lactate	1.6 mmol/L

Which is the most appropriate initial intervention?

(Please select 1 option)

○	Intravenous infusion of 1.26% sodium bicarbonate and potassium replacement
○	Potassium supplementation via a central venous catheter
○	Intravenous sliding scale insulin infusion
○	Rehydration with 0.9% sodium chloride and correction of blood glucose
○	Encourage patient to breathe into a paper bag



● Intravenous infusion of 1.26% sodium bicarbonate and potassium replacement

○ Potassium supplementation via a central venous catheter

○ Intravenous sliding scale insulin infusion

○ Rehydration with 0.9% sodium chloride and correction of blood glucose

○ Encourage patient to breathe into a paper bag

Key Learning Points

Critical Care, Neurology

- Hyperchloraemic metabolic acidosis is a documented complication of neobladder formation.

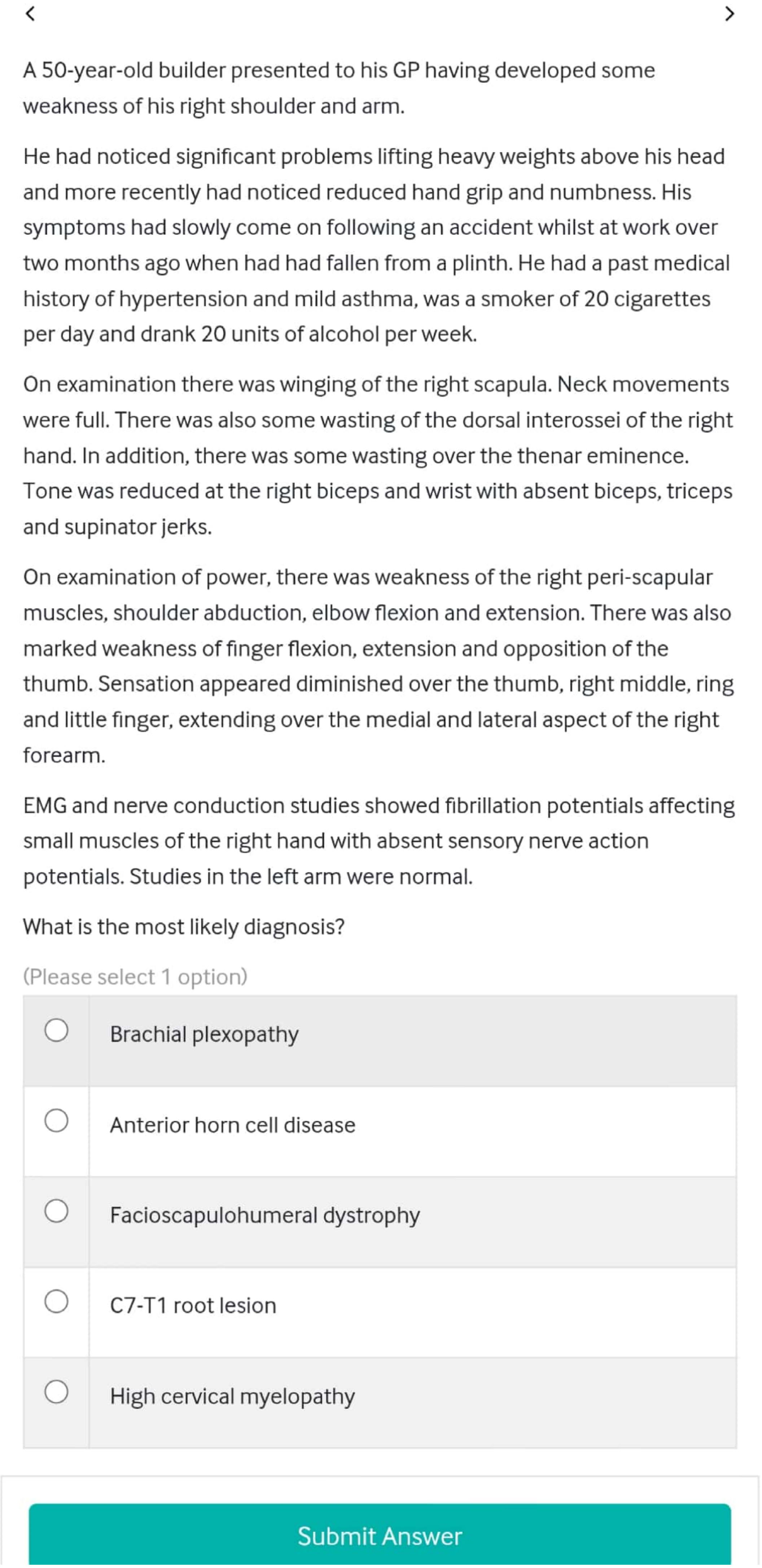
Explanation

The patient has a hyperchoraemic metabolic acidosis with respiratory compensation.

There is also a mild hypernatraemia and hypokalaemia. Lactate concentrations are normal which suggests that the acidosis is not secondary to organ hypoperfusion.

Hyperchloraemic metabolic acidosis is a documented complication of neobladder formation. However, it usually improves with time and is mild. Severe and persistent metabolic acidosis may manifest when patients undergo further surgery for other reasons, as is the case in this patient. Neobladder formation following radical cystectomy or cystoprostatectomy is becoming increasingly more common, and medical staff treating patients with neobladders should recognise and treat metabolic acidosis with intravenous fluids and bicarbonate. Associated electrolyte abnormalities may include hypokalemia, hypocalcaemia, and hypomagnesaemia. Hypokalemia is caused by both intestinal (secretion) and renal losses. This depletion of potassium can be exacerbated by the correction of acidosis.

Rehydrating the patient with 0.9% N. saline is likely to exacerbate the hyperchloraemic state. Potassium supplementation alone via a central venous catheter will not be sufficient treatment. Encouraging the patient to breathe into a paper bag might be a treatment of an acute respiratory alkalosis but inappropriate in this patient. The patient has hyperglycaemia secondary to the metabolic stress response and not ketoacidosis.



A 50-year-old builder presented to his GP having developed some weakness of his right shoulder and arm.

He had noticed significant problems lifting heavy weights above his head and more recently had noticed reduced hand grip and numbness. His symptoms had slowly come on following an accident whilst at work over two months ago when had had fallen from a plinth. He had a past medical history of hypertension and mild asthma, was a smoker of 20 cigarettes per day and drank 20 units of alcohol per week.

On examination there was winging of the right scapula. Neck movements were full. There was also some wasting of the dorsal interossei of the right hand. In addition, there was some wasting over the thenar eminence. Tone was reduced at the right biceps and wrist with absent biceps, triceps and supinator jerks.

On examination of power, there was weakness of the right peri-scapular muscles, shoulder abduction, elbow flexion and extension. There was also marked weakness of finger flexion, extension and opposition of the thumb. Sensation appeared diminished over the thumb, right middle, ring and little finger, extending over the medial and lateral aspect of the right forearm.

EMG and nerve conduction studies showed fibrillation potentials affecting small muscles of the right hand with absent sensory nerve action potentials. Studies in the left arm were normal.

What is the most likely diagnosis?

(Please select 1 option)

○ Brachial plexopathy

○ Anterior horn cell disease

○ Facioscapulohumeral dystrophy

○ C7-T1 root lesion

○ High cervical myelopathy

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● Brachial plexopathy **Correct**

○ Anterior horn cell disease

○ Facioscapulohumeral dystrophy

○ C7-T1 root lesion

○ High cervical myelopathy

Key Learning Points

Neurology

- Brachial plexus injury is common following trauma and leads to motor and sensory disturbance across multiple dermatomes and myotomes

Explanation

This patient has a history and clinical findings in keeping with a diagnosis of traumatic brachial plexopathy with C5-T1 root involvement. He has winged scapula and weakness affecting elbow flexion/extension and small muscles of the hand and sensory loss affecting dermatomes C6-T1.

The lesion is most likely in the plexus rather than nerve root, as the nerve conduction studies confirm absent sensory nerve action potentials, pointing to a lesion that is distal to the dorsal root ganglion.

There is nothing in the clinical history to suggest anterior horn cell disease or a muscle dystrophy.

A high cervical myelopathy would lead to upper motor neurone signs in the upper limb with respiratory involvement.

Next Question

A 32-year-old woman was referred to neurology clinic with a progressive history over a number of years of unsteadiness on her legs. She had noticed a worsening of balance recently, which had resulted in a number of falls. She had also noticed slurring of speech and had recently developed a bilateral hand tremor. She complained that her feet were cold and numb.

She had recently been diagnosed with diabetes and had been commenced on metformin. Otherwise she had no other medical problems and was not on any other regular medication. She was a non-smoker and drank 25 units of alcohol/week. Her mother had suffered from diabetes and a heart condition.

On examination she was pale with a blood pressure of 140/78 mmHg, pulse of 78/minute and normal heart sounds. Fundoscopy revealed bilateral optic atrophy. Ocular movements were full, however there was coarse horizontal nystagmus. She had a marked dysarthria, however the rest of the cranial nerves were entirely normal.

Examination of the upper limb revealed bilateral intention tremor with poor finger to nose testing on both sides. Tone, power, reflexes and sensation appeared normal. Examination of the lower limb revealed bilateral pes cavus. Tone was mildly increased in both legs. There was no apparent weakness, however knee and ankle reflexes were bilaterally absent, and both plantar responses were extensor. There was some loss of sensation in the legs, with vibration felt at both knees and impaired joint position sense. The rest of the sensory modalities were intact.

Investigations revealed:

Haemoglobin	132 g/L	(115 - 165)
Mean cell volume	108 fL	(80 - 96)
White cell count	5.7 ×10^9/L	(4 - 11)
Platelets	285 ×10^9/L	(150 - 400)
Serum sodium	136 mmol/L	(137 - 144)
Serum potassium	4.1 mmol/L	(3.5 - 4.9)
Serum urea	5.4 mmol/L	(2.5 - 7.5)
Serum creatinine	107 µmol/L	(60 - 110)
Fasting plasma glucose	8.9 mmol/L	(3 - 6)

A lumbar puncture was performed. Analysis of the cerebrospinal fluid showed:

CSF white cell count	3 per ml	(<5)
CSF red cell count	2 per ml	(<5)
CSF glucose	4.4 mmol/L	(3.3 - 4.4)
CSF protein	0.35 g/L	(0.15 - 0.45)

CSF oligoclonal bands negative. Serum oligoclonal bands negative.

What is the most likely diagnosis in this patient?

(Please select 1 option)

○	Subacute combined degeneration of the cord
○	Friedreich's ataxia
○	Hypothyroidism
○	Multiple sclerosis
○	Alcohol abuse

Submit Answer

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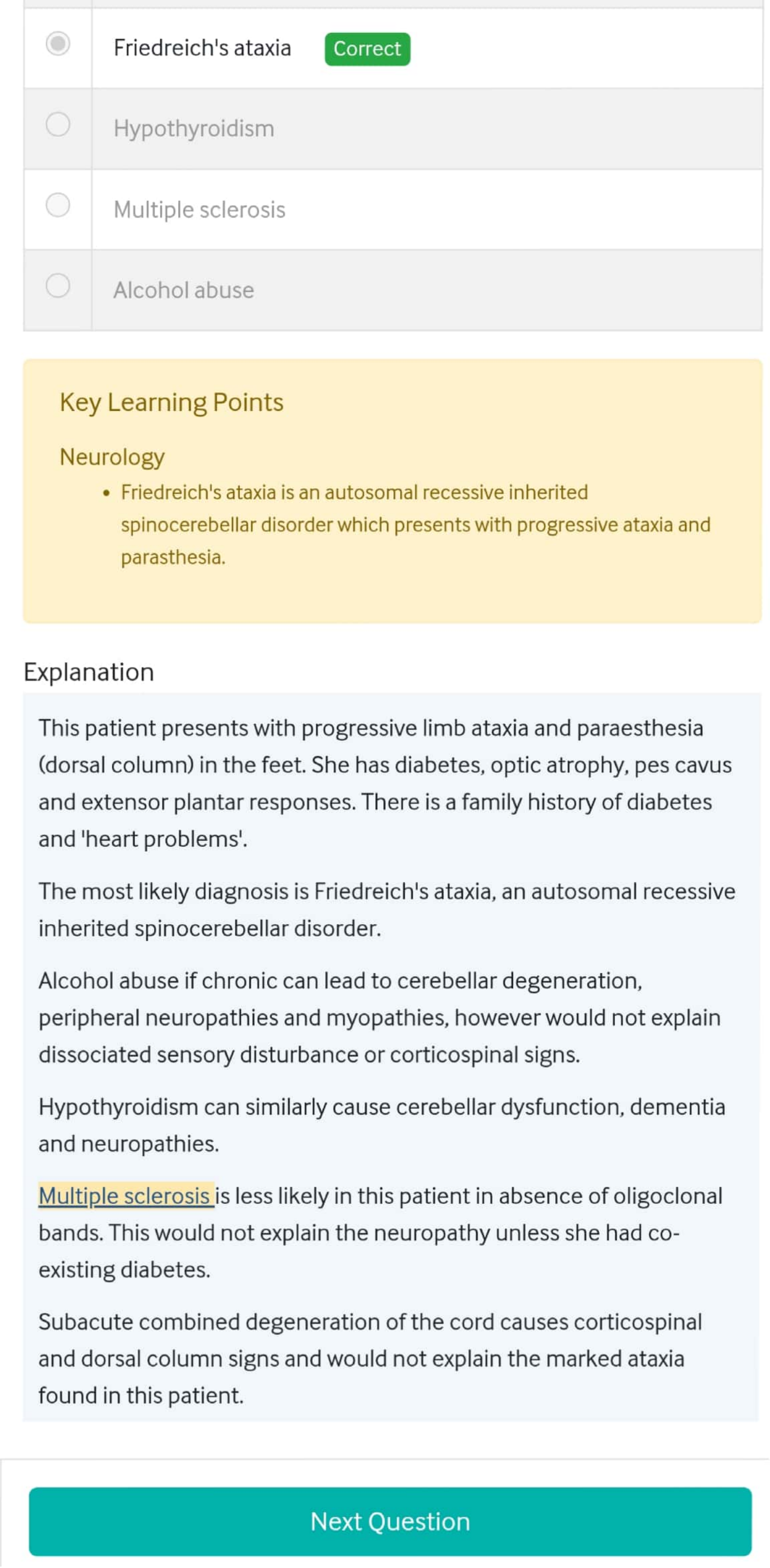

● Friedreich's ataxia Correct

○ Hypothyroidism

○ Multiple sclerosis

○ Alcohol abuse

Key Learning Points

Neurology

- Friedreich's ataxia is an autosomal recessive inherited spinocerebellar disorder which presents with progressive ataxia and parasthesia.

Explanation

This patient presents with progressive limb ataxia and paraesthesia (dorsal column) in the feet. She has diabetes, optic atrophy, pes cavus and extensor plantar responses. There is a family history of diabetes and 'heart problems'.

The most likely diagnosis is Friedreich's ataxia, an autosomal recessive inherited spinocerebellar disorder.

Alcohol abuse if chronic can lead to cerebellar degeneration, peripheral neuropathies and myopathies, however would not explain dissociated sensory disturbance or corticospinal signs.

Hypothyroidism can similarly cause cerebellar dysfunction, dementia and neuropathies.

Multiple sclerosis is less likely in this patient in absence of oligoclonal bands. This would not explain the neuropathy unless she had co-existing diabetes.

Subacute combined degeneration of the cord causes corticospinal and dorsal column signs and would not explain the marked ataxia found in this patient.

Question 113

Question 113 of 133

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A 17-year-old male presents with a one week history of progressive lower limb weakness, difficulty swallowing, and shortness of breath. He denied any history of trauma.

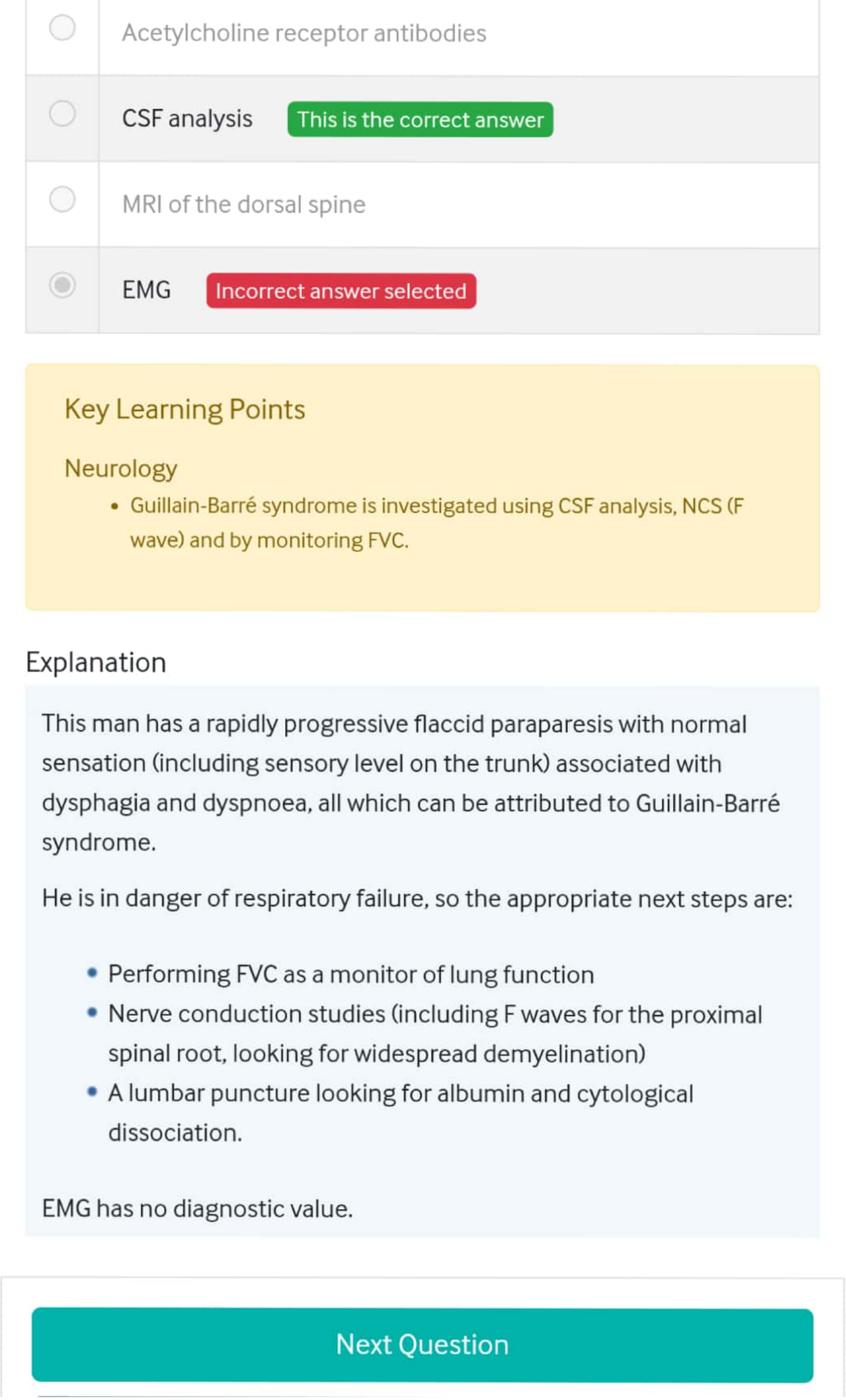
Examination reveals absent ankle and knee jerks with non-responsive planters and hypotonic weakness of grade 2 in both lower limbs. Sensation was intact.

Which of the following are the most relevant next steps for this man?

(Please select 1 option)

- ○ Tensilon test
- ○ Acetylcholine receptor antibodies
- ○ CSF analysis
- ○ MRI of the dorsal spine
- ○ EMG

Submit Answer



○ Acetylcholine receptor antibodies

○ CSF analysis This is the correct answer

○ MRI of the dorsal spine

● EMG Incorrect answer selected

Key Learning Points

Neurology

- Guillain-Barré syndrome is investigated using CSF analysis, NCS (F wave) and by monitoring FVC.

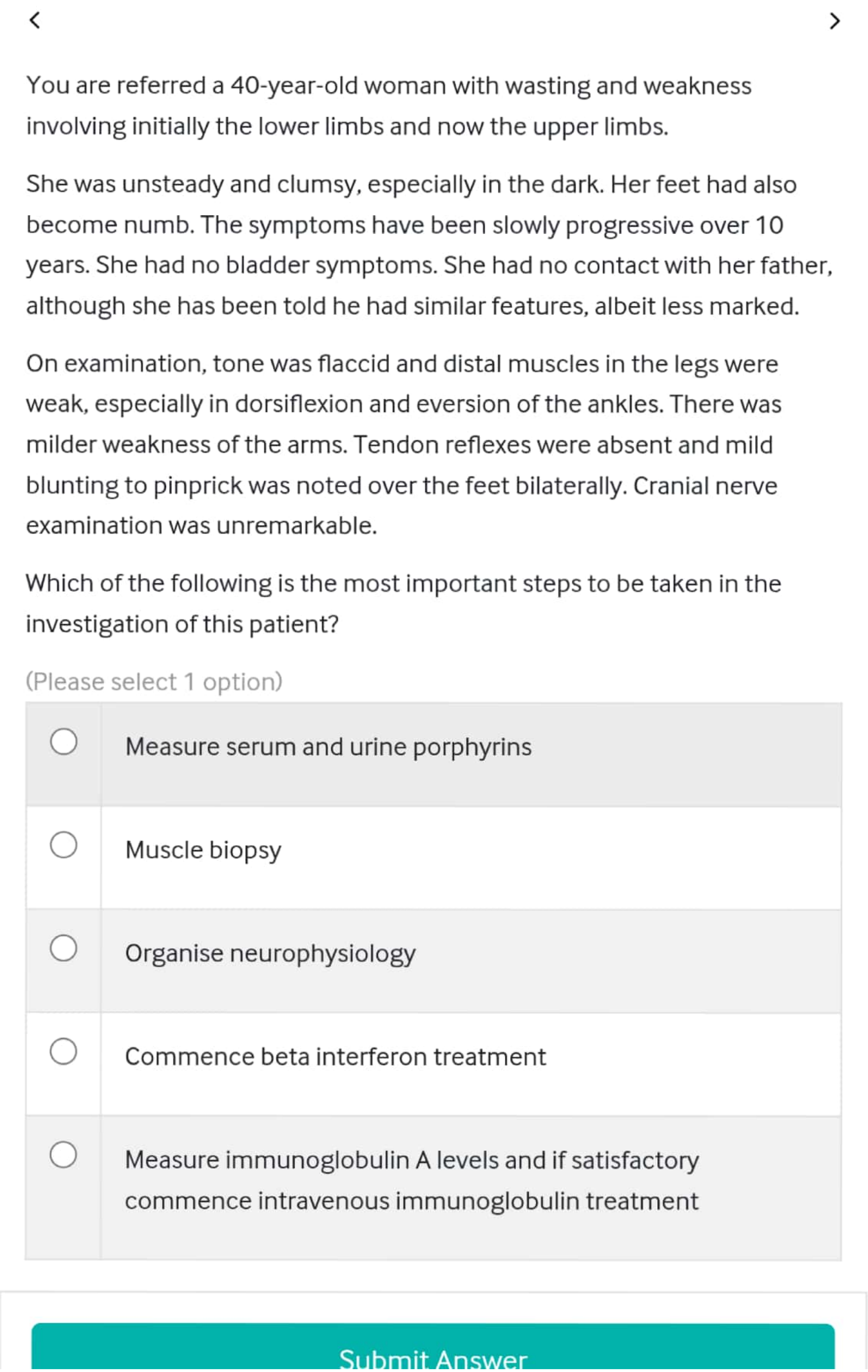
Explanation

This man has a rapidly progressive flaccid paraparesis with normal sensation (including sensory level on the trunk) associated with dysphagia and dyspnoea, all which can be attributed to Guillain-Barré syndrome.

He is in danger of respiratory failure, so the appropriate next steps are:

- Performing FVC as a monitor of lung function
- Nerve conduction studies (including F waves for the proximal spinal root, looking for widespread demyelination)
- A lumbar puncture looking for albumin and cytological dissociation.

EMG has no diagnostic value.



You are referred a 40-year-old woman with wasting and weakness involving initially the lower limbs and now the upper limbs.

She was unsteady and clumsy, especially in the dark. Her feet had also become numb. The symptoms have been slowly progressive over 10 years. She had no bladder symptoms. She had no contact with her father, although she has been told he had similar features, albeit less marked.

On examination, tone was flaccid and distal muscles in the legs were weak, especially in dorsiflexion and eversion of the ankles. There was milder weakness of the arms. Tendon reflexes were absent and mild blunting to pinprick was noted over the feet bilaterally. Cranial nerve examination was unremarkable.

Which of the following is the most important steps to be taken in the investigation of this patient?

(Please select 1 option)

- ○ Measure serum and urine porphyrins
- ○ Muscle biopsy
- ○ Organise neurophysiology
- ○ Commence beta interferon treatment
- ○ Measure immunoglobulin A levels and if satisfactory commence intravenous immunoglobulin treatment

○ Organise neurophysiology **This is the correct answer**

○ Commence beta interferon treatment

● Measure immunoglobulin A levels and if satisfactory commence intravenous immunoglobulin treatment **Incorrect answer selected**

Key Learning Points

Neurology

- HSMN has various types. Genetic testing usually reveals the diagnosis and EMG and NCS help distinguish between the types.

Explanation

A logical approach to the assessment of such patients is essential and can be organised into a number of basic questions. The neurologist faced with a patient with a neuropathy has to deal with literally thousands of possible causes, many extremely uncommon, and these can only be simplified by defining the neuropathy by other features that lead to the select few of most likely diagnoses.

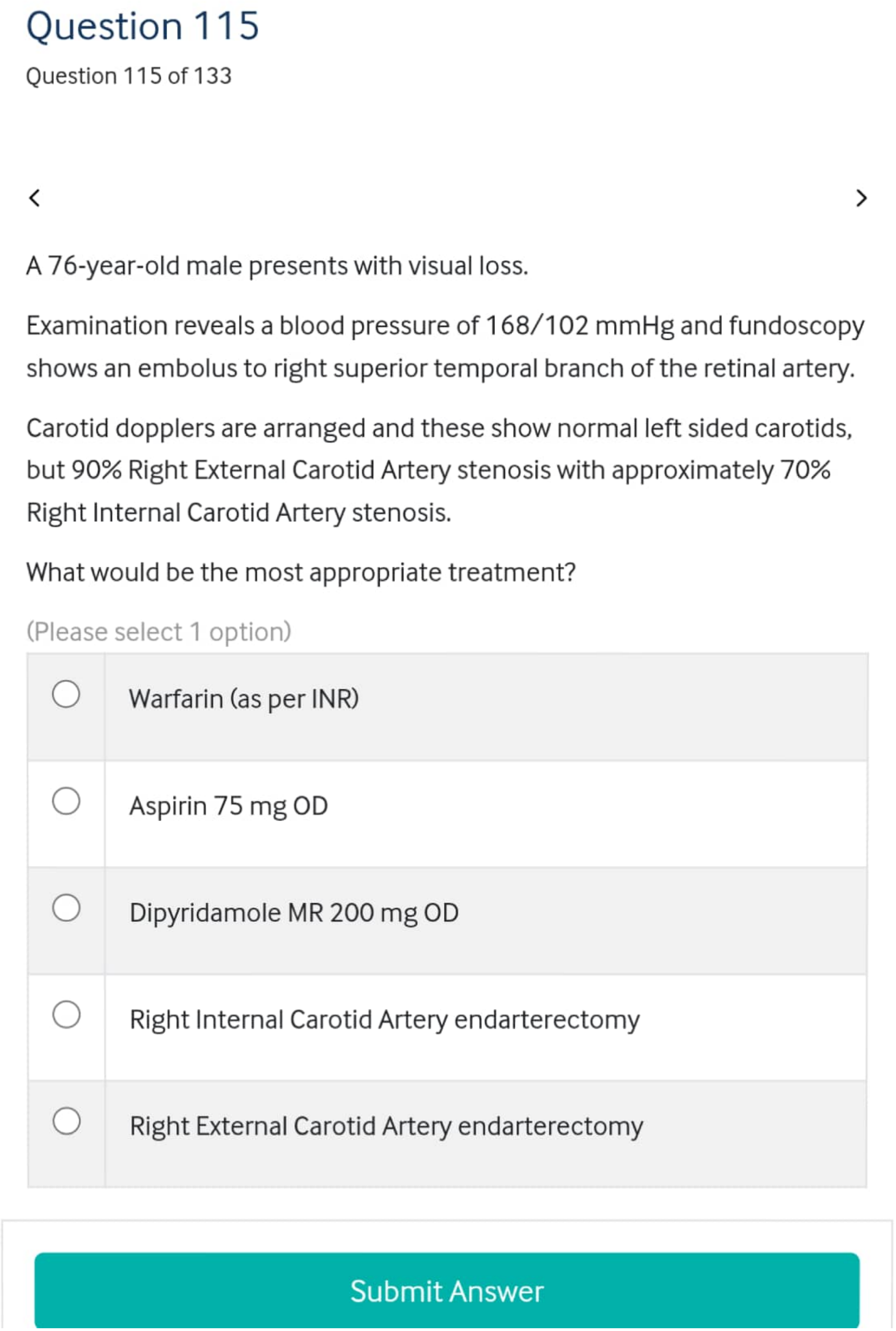
Mixed motor and sensory symptoms, slowly progressing initially in the lower limbs and then to the upper limbs, together with a family history suggests a diagnosis of hereditary motor sensory neuropathy (HMSN).

Electromyography and nerve conduction studies may distinguish between the demyelinating (type 1) and axonal (type 2) forms.

Genetic testing is available for certain types of HMSN. Genetic testing usually reveals the diagnosis and EMG and NCS help distinguish between the types.

Then the next step is neurophysiology - this will give information about whether this could be a central or root disorder and point to demyelination.
It is less invasive than muscle biopsy (or nerve biopsy) and we do know sufficient information to commence treatment with beta interferon.
Serum IgA levels may help, but there are many diagnoses still to be excluded and this test does not have enough discriminatory value.

Next Question



Question 115

Question 115 of 133

A 76-year-old male presents with visual loss.

Examination reveals a blood pressure of 168/102 mmHg and fundoscopy shows an embolus to right superior temporal branch of the retinal artery.

Carotid dopplers are arranged and these show normal left sided carotids, but 90% Right External Carotid Artery stenosis with approximately 70% Right Internal Carotid Artery stenosis.

What would be the most appropriate treatment?

(Please select 1 option)

- ○ Warfarin (as per INR)
- ○ Aspirin 75 mg OD
- ○ Dipyridamole MR 200 mg OD
- ○ Right Internal Carotid Artery endarterectomy
- ○ Right External Carotid Artery endarterectomy

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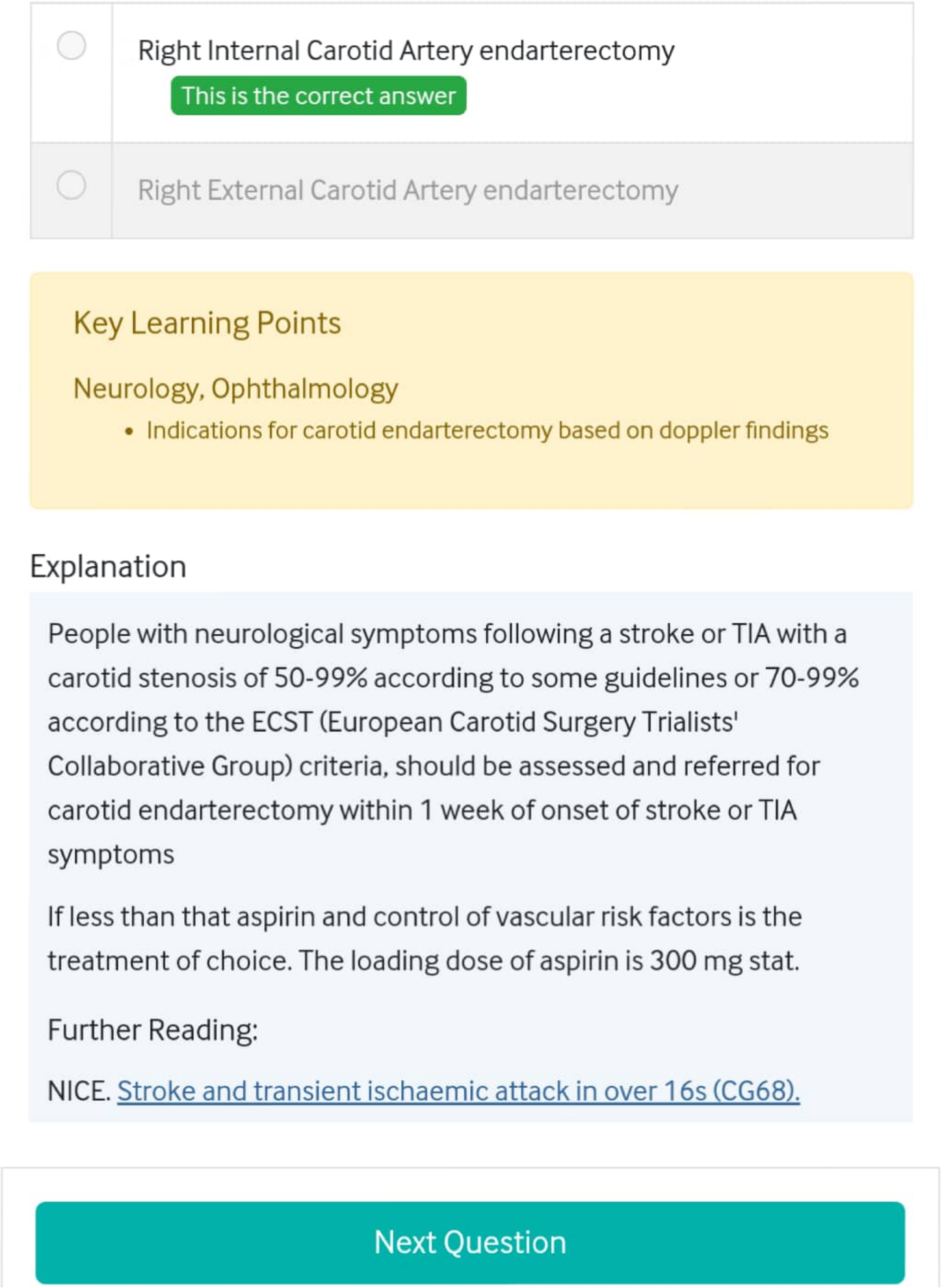


○ Right Internal Carotid Artery endarterectomy

This is the correct answer

○ Right External Carotid Artery endarterectomy

Key Learning Points

Neurology, Ophthalmology

- Indications for carotid endarterectomy based on doppler findings

Explanation

People with neurological symptoms following a stroke or TIA with a carotid stenosis of 50-99% according to some guidelines or 70-99% according to the ECST (European Carotid Surgery Trialists' Collaborative Group) criteria, should be assessed and referred for carotid endarterectomy within 1 week of onset of stroke or TIA symptoms

If less than that aspirin and control of vascular risk factors is the treatment of choice. The loading dose of aspirin is 300 mg stat.

Further Reading:

NICE. Stroke and transient ischaemic attack in over 16s (CG68).

CamScanner الممسوحة ضوئيا بـ

A 45-year-old labourer is referred with a three month history of tingling and paraesthesia of the left hand.

On two occasions he dropped his cup of tea but put these down to a bit of clumsiness. His symptoms tend to be more noticeable in the evenings when he is resting or watching TV. He denies any muscle twitching.

Bladder and bowel function are reported as normal. Past medical history is unremarkable and he has not been involved in any accidents recently. His job means that he often has to use vibrating tools and do heavy manual lifting.

On examination, he has no obvious loss of muscle bulk in his upper limbs. Tone is normal. Power appears normal in all muscle groups. Apart from an absent triceps reflex on the left, his reflexes are all present and symmetrical. Sensory examination reveals loss of pinprick sensation on the palmar aspect of the index, middle and ring fingers of the left hand. Tapping of the palmar aspect of the wrist fails to elicit any tingling sensations in the hand distally. Joint position sense and vibration sense are both normal. Cranial nerve examination is unremarkable.

What is the most likely diagnosis?

(Please select 1 option)

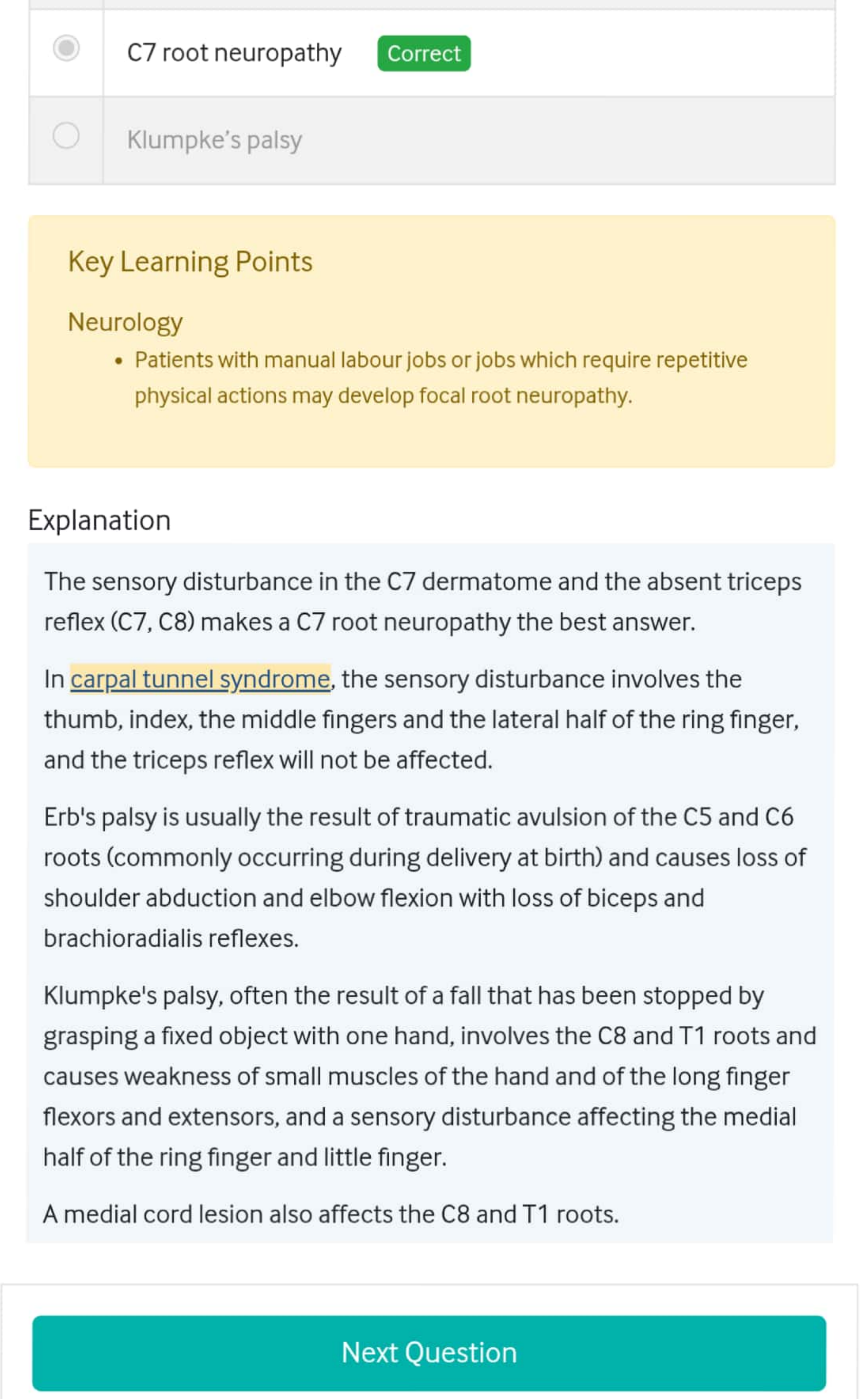
○ Carpal tunnel syndrome

○ Medial cord lesion

○ Erb’s palsy

○ C7 root neuropathy

○ Klumpke’s palsy



● C7 root neuropathy **Correct**

○ Klumpke's palsy

Key Learning Points

Neurology

- Patients with manual labour jobs or jobs which require repetitive physical actions may develop focal root neuropathy.

Explanation

The sensory disturbance in the C7 dermatome and the absent triceps reflex (C7, C8) makes a C7 root neuropathy the best answer.

In carpal tunnel syndrome, the sensory disturbance involves the thumb, index, the middle fingers and the lateral half of the ring finger, and the triceps reflex will not be affected.

Erb's palsy is usually the result of traumatic avulsion of the C5 and C6 roots (commonly occurring during delivery at birth) and causes loss of shoulder abduction and elbow flexion with loss of biceps and brachioradialis reflexes.

Klumpke's palsy, often the result of a fall that has been stopped by grasping a fixed object with one hand, involves the C8 and T1 roots and causes weakness of small muscles of the hand and of the long finger flexors and extensors, and a sensory disturbance affecting the medial half of the ring finger and little finger.

A medial cord lesion also affects the C8 and T1 roots.

A 58-year-old woman was referred to a neurologist with episodes of loss of consciousness.

She had already been seen by a cardiologist and had been investigated with an echocardiogram and a fitted loop recorder, both of which had been entirely normal.

She described these events as initially starting with an occipital headache, which abates. She then started to feel dizzy, things started to spin and she developed double vision and dysarthria. On some of these occasions she had completely blacked out and has been found to twitch all four limbs. When coming round from an event it took her a minute or two to become orientated and resume her activities. She had had one episode of incontinence. Her last episode was whilst at the hairdressers having her hair washed.

She had a past history of hypertension treated with bendroflumethiazide 2.5 mg.

On examination her blood pressure was 162/89 mmHg (lying) and 157/85 mmHg (standing), pulse was 89/min and regular, heart sounds were normal and chest was clear. There was a right carotid bruit. Her blood glucose-monitoring test was 7.9. There were no abnormalities found on examining cranial nerves and peripheral nervous system examination appeared normal.

CT scan is normal.

Blood tests showed:

Sodium	140 mmol/L	(137-144)
Potassium	3.4 mmol/L	(3.5-4.9)
Urea	5.8 mmol/L	(2.5-7.5)
Creatinine	101 µmol/L	(60-110)
Cholesterol	6.9 mmol/L	(<5.2)
Glucose	7.7 mmol/L	(3.0-6.0)

Given the history and results so far, what is the most likely diagnosis in this patient?

(Please select 1 option)

○ Carotid artery stenosis and multiple TIAs

○ Vertebrobasilar insufficiency

○ Postural hypotension

○ Occipital lobe seizures

○ Basilar migraine

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○ Vertebrobasilar insufficiency **This is the correct answer**

○ Postural hypotension

○ Occipital lobe seizures

○ Basilar migraine

Key Learning Points

Neurology

- Brainstem features include diplopia, dysarthria and dizziness.

Explanation

This patient presents with short episodes lasting minutes of loss of consciousness associated with brainstem features (diplopia, dysarthria and dizziness).

They are not posturally related, and on some occasions have been associated with reflex anoxic seizures.

There does not appear to be any history of confusion following these attacks or any precipitating factors. Hyperextension of the neck however can precipitate loss of consciousness, which may have occurred in the hairdressers.

These features are compatible with a history of vertebrobasilar insufficiency. She also appears to have several vascular risk factors including hypertension, hypercholesterolaemia and probable glucose intolerance.

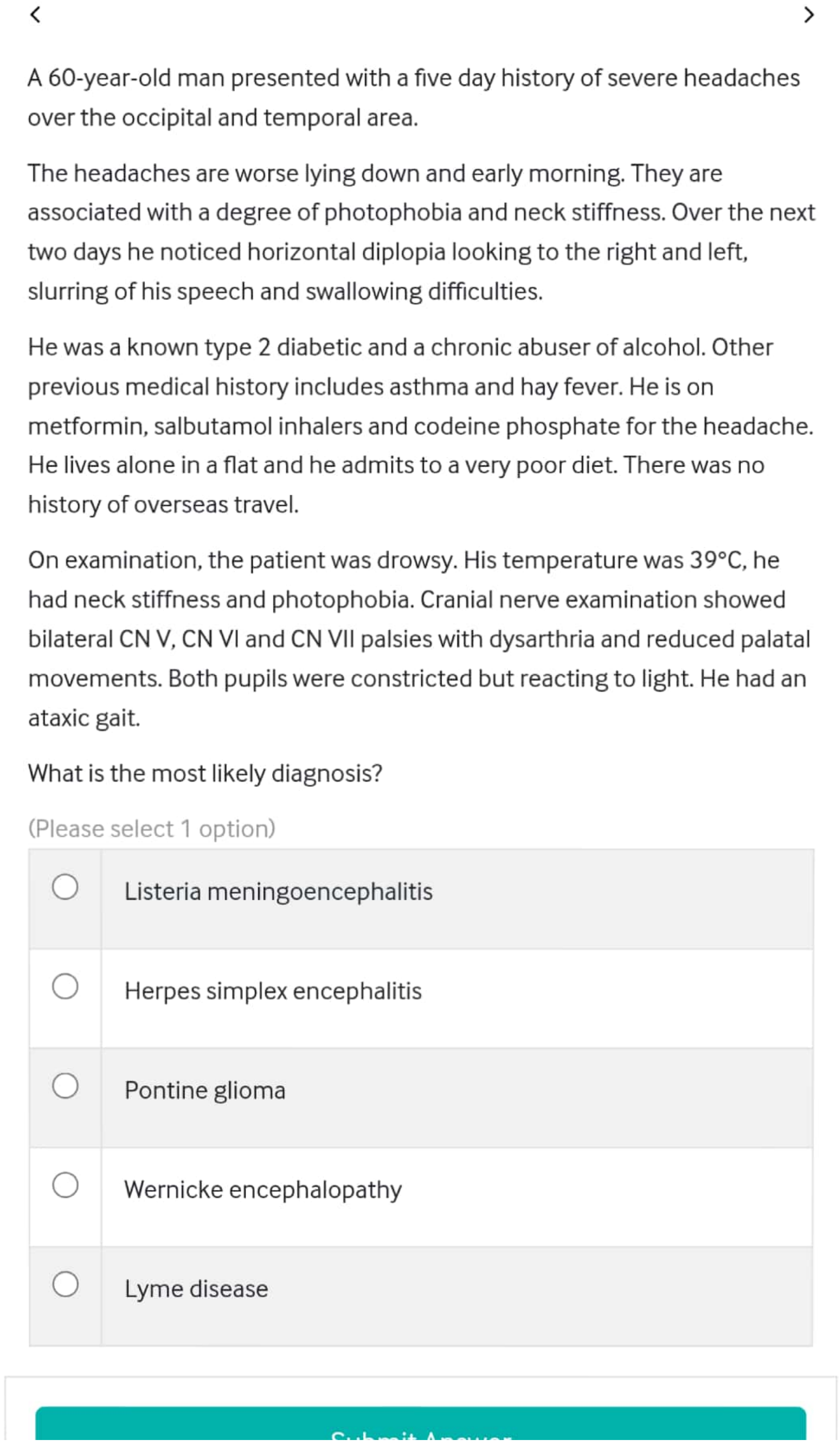
In occipital lobe seizures one would expect a history of visual field disturbance or elementary visual hallucinations.

Carotid artery territory ischaemia would not cause loss of consciousness.

Postural hypotension is unlikely in view of the non-postural nature of the attacks and lying and standing BP readings.

Basilar migraine is a possibility, however this patient has no prior history of headaches and it is unusual for neurological deficits to continue after the onset of headache.

Next Question



A 60-year-old man presented with a five day history of severe headaches over the occipital and temporal area.

The headaches are worse lying down and early morning. They are associated with a degree of photophobia and neck stiffness. Over the next two days he noticed horizontal diplopia looking to the right and left, slurring of his speech and swallowing difficulties.

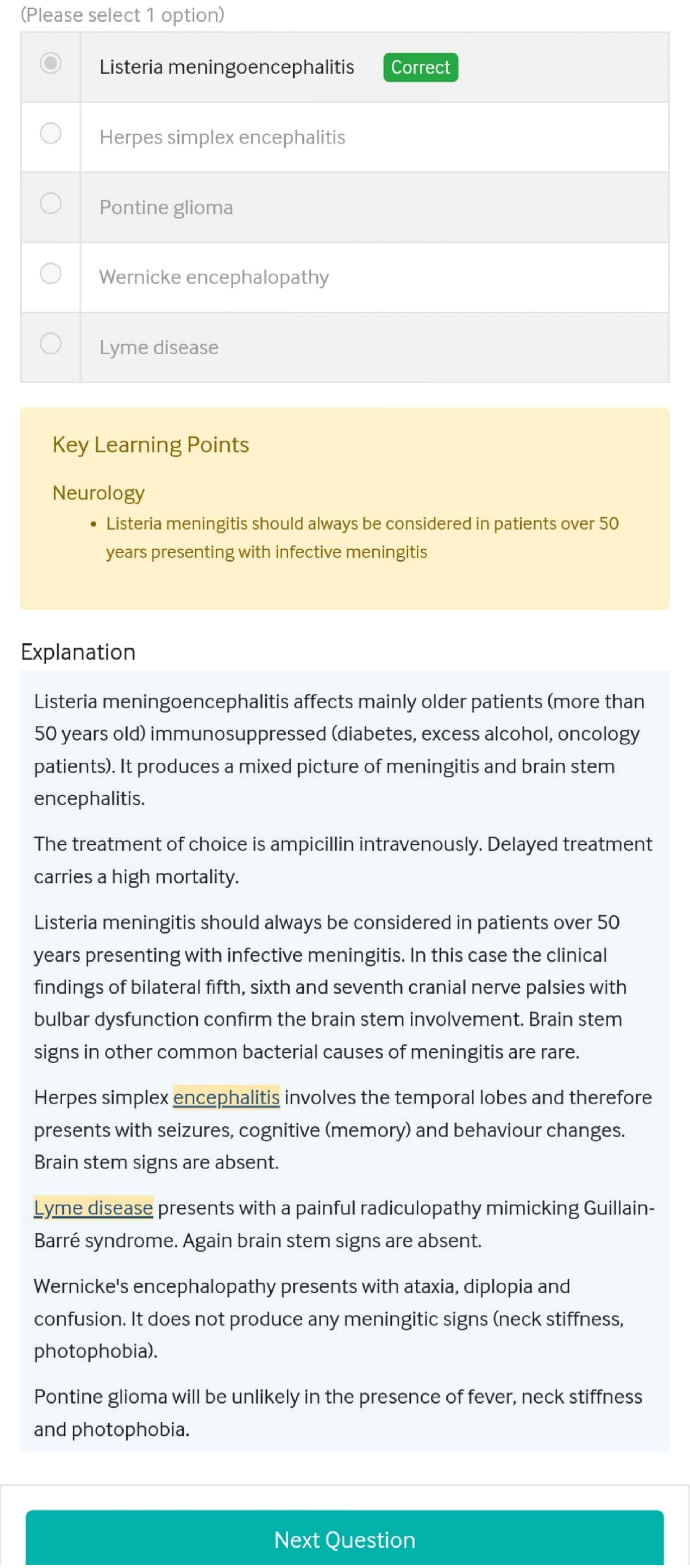
He was a known type 2 diabetic and a chronic abuser of alcohol. Other previous medical history includes asthma and hay fever. He is on metformin, salbutamol inhalers and codeine phosphate for the headache. He lives alone in a flat and he admits to a very poor diet. There was no history of overseas travel.

On examination, the patient was drowsy. His temperature was 39°C, he had neck stiffness and photophobia. Cranial nerve examination showed bilateral CN V, CN VI and CN VII palsies with dysarthria and reduced palatal movements. Both pupils were constricted but reacting to light. He had an ataxic gait.

What is the most likely diagnosis?

(Please select 1 option)

- ○ Listeria meningoencephalitis
- ○ Herpes simplex encephalitis
- ○ Pontine glioma
- ○ Wernicke encephalopathy
- ○ Lyme disease



(Please select 1 option)

- ● Listeria meningoencephalitis **Correct**
- ○ Herpes simplex encephalitis
- ○ Pontine glioma
- ○ Wernicke encephalopathy
- ○ Lyme disease

Key Learning Points

Neurology

- Listeria meningitis should always be considered in patients over 50 years presenting with infective meningitis

Explanation

Listeria meningoencephalitis affects mainly older patients (more than 50 years old) immunosuppressed (diabetes, excess alcohol, oncology patients). It produces a mixed picture of meningitis and brain stem encephalitis.

The treatment of choice is ampicillin intravenously. Delayed treatment carries a high mortality.

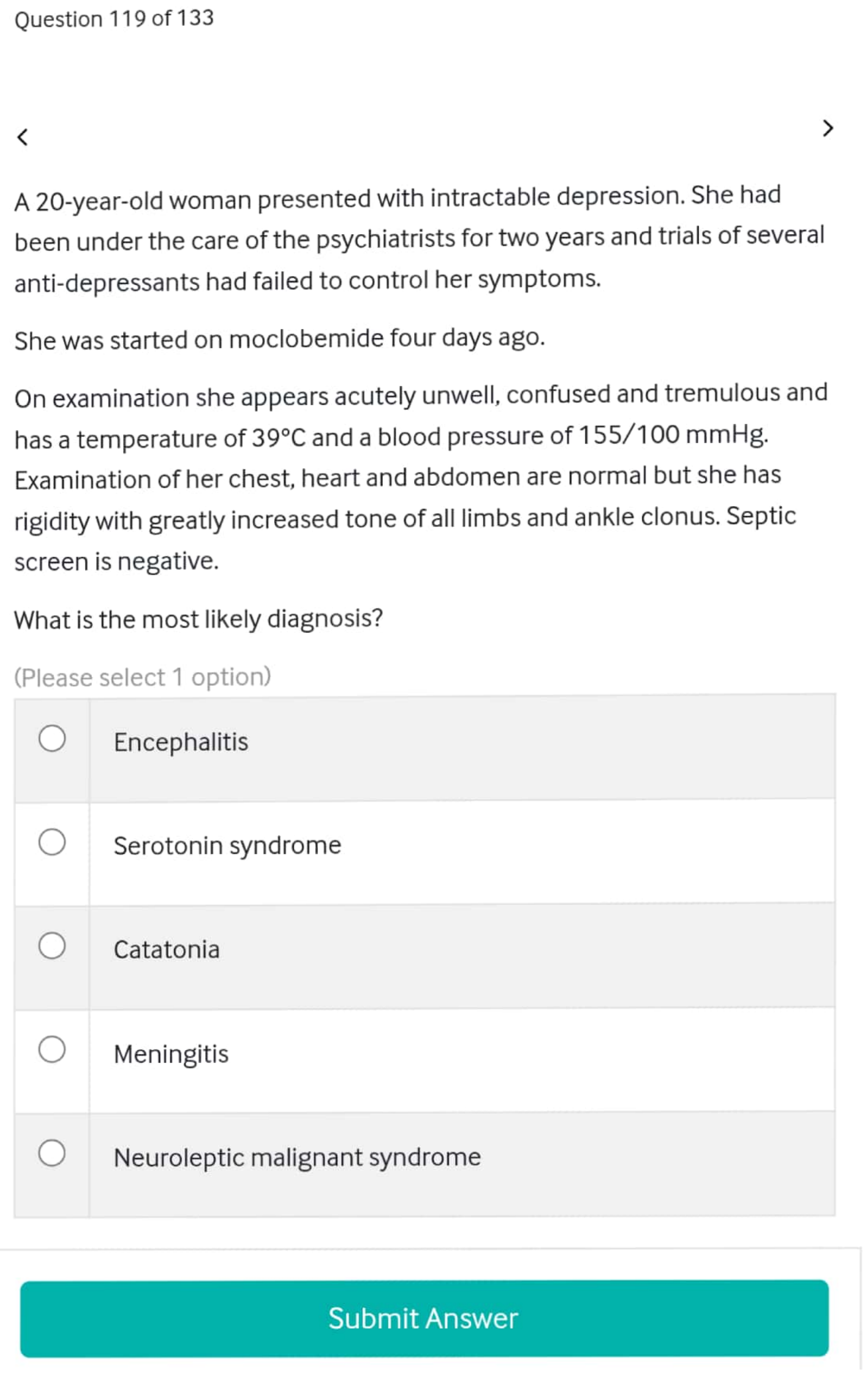
Listeria meningitis should always be considered in patients over 50 years presenting with infective meningitis. In this case the clinical findings of bilateral fifth, sixth and seventh cranial nerve palsies with bulbar dysfunction confirm the brain stem involvement. Brain stem signs in other common bacterial causes of meningitis are rare.

Herpes simplex encephalitis involves the temporal lobes and therefore presents with seizures, cognitive (memory) and behaviour changes. Brain stem signs are absent.

Lyme disease presents with a painful radiculopathy mimicking Guillain-Barré syndrome. Again brain stem signs are absent.

Wernicke's encephalopathy presents with ataxia, diplopia and confusion. It does not produce any meningitic signs (neck stiffness, photophobia).

Pontine glioma will be unlikely in the presence of fever, neck stiffness and photophobia.



Question 119 of 133

A 20-year-old woman presented with intractable depression. She had been under the care of the psychiatrists for two years and trials of several anti-depressants had failed to control her symptoms.

She was started on moclobemide four days ago.

On examination she appears acutely unwell, confused and tremulous and has a temperature of 39°C and a blood pressure of 155/100 mmHg. Examination of her chest, heart and abdomen are normal but she has rigidity with greatly increased tone of all limbs and ankle clonus. Septic screen is negative.

What is the most likely diagnosis?

(Please select 1 option)

○ Encephalitis

○ Serotonin syndrome

○ Catatonia

○ Meningitis

○ Neuroleptic malignant syndrome

● Serotonin syndrome **Correct**

○ Catatonia

○ Meningitis

○ Neuroleptic malignant syndrome

Key Learning Points

Neurology, Psychiatry

- Consider serotonin syndrome in a patient who suddenly develops neuromuscular hyperactivity, fever, confusion and muscle rigidity.

Explanation

The most likely diagnosis is serotonin syndrome.

Moclobemide is indicated for major depression and social phobia.

It is a reversible monoamine oxidase inhibitor (MAOI). It is well known to cause serotonin syndrome. The syndrome has many common features with neuroleptic malignant syndrome.

Serotonin syndrome is often misdiagnosed as NMS, but the two can easily be distinguished on the basis of history, examination findings, and clinical course. NMS develops over days to weeks, whereas serotonin syndrome develops over 24 hours. Serotonin syndrome is characterized by neuromuscular hyperreactivity (tremor, hyperreflexia, myoclonus), while NMS involves sluggish neuromuscular responses (rigidity, bradyreflexia). Hyperreflexia and myoclonus are rare in NMS. Also, resolution of NMS typically requires an average of nine days, compared with less than 24 hours (usually) for resolution of serotonin syndrome

Hyperthermia, altered mental status, muscle rigidity, leukocytosis, elevated creatine phosphokinase, elevated hepatic transaminases, and metabolic acidosis are seen in severe cases of both conditions, which highlights the necessity of a thorough history and physical examination.

Next Question

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Question 120

Question 120 of 133

A 17-year-old girl presents following a collapse in the supermarket.

She describes feeling hot and sweaty, with blurred vision before she passed out. Her mother states she was unresponsive and that there was twitching of her limbs for twenty seconds before she came round.

After a few minutes she was orientated but described feeling sick. She did not bite her tongue but was incontinent of urine. She has had three similar episodes in the past. Her older brother has epilepsy. An ECG showed normal sinus rhythm.

What investigation is most likely to lead to the correct diagnosis?

(Please select 1 option)

- ○ MRI scan of the head
- ○ CT scan of the head
- ○ Holter monitor
- ○ Electroencephalography
- ○ Tilt table test

Submit Answer

○ Tilt table test This is the correct answer

Key Learning Points

Neurology

- Patients with syncope can commonly have jerking of the limbs when they are unconscious and this does not mean they have had a seizure. Tilt table testing is useful to support the diagnosis of vasovagal syncope.

Explanation

The most likely diagnosis is vasovagal syncope with characteristic warning symptoms of feeling hot and blurring of vision with quick recovery.

Patients with syncope can commonly have jerking of the limbs when they are unconscious and this does not mean they have had a seizure. Tilt table testing is useful to support the diagnosis of vasovagal syncope.

CT head, EEG, MRI are not suitable as this girl has not had a seizure.

Holter monitoring can be used to investigate recurrent or atypical episodes of syncope but in the presence of a normal ECG is unlikely to provide a diagnosis.

Next Question

Question 121 of 133

Core Questions

‹ ›

A 34-year-old female patient presented with a four week history of bifrontal headache, decreasing visual acuity and walking difficulties. There was no significant medical history.

On examination of cranial nerves there was bilateral VIth nerve palsy and fundoscopy revealed bilateral papilloedema. Visual acuity was reduced and gait was ataxic.

Routine blood count revealed an elevated platelet count of 867 $\times 10^9$/L. Haemoglobin, haematocrit, white cell count, iron store and inflammatory markers were all normal. MRI brain showed cerebral venous sinus thrombosis with a small cerebellar haemorrhage.

What is the next best step in management of this patient?

(Please select 1 option)

- ○ Intravenous unfractionated heparin
- ○ Thrombolysis
- ○ Combination of plasma exchange and corticosteroids
- ○ Aspirin only
- ○ Hydroxycarbamide only

Submit Answer

● Intravenous unfractionated heparin **Correct**

○ Thrombolysis

○ Combination of plasma exchange and corticosteroids

○ Aspirin only

○ Hydroxycarbamide only

Key Learning Points

Neurology

- Patients with cerebral venous sinus thrombosis (CVST) require IV heparin infusion which should be continued even in case of haeorrhagic transformation.

Explanation

This clinical scenario describes the case of haemorrhagic cerebral venous sinus thrombosis (CVST) due to essential thrombocytosis.

Essential thrombocytosis (ET) is a chronic myeloproliferative disorder associated with sustained megakaryocyte proliferation, increasing the number of circulating platelets (above 450×10^9/L). It is associated with thrombosis and bleeding.

Thrombotic events may include:

- Stroke
- Transient ischaemic attacks (TIAs)
- Retinal artery or venous occlusions
- Coronary artery ischaemia
- Myocardial infarction (MI)
- Pulmonary embolism
- Hepatic or portal vein thrombosis
- Deep vein thrombosis (DVT), and
- Digital ischaemia.[2]

Neurological complications of ET may derive from:[1,3,4]

1. Major artery thrombosis (TIA, stroke, spinal infarct)
2. Vein thrombosis (CVST and high pressure headache, reduced consciousness, focal neurology)
3. Microangiopathic complications (amaurosis fugax, reduced visual acuity, erythromelalgia and Raynaud's phenomenon).

Intracranial haemorrhage is a serious bleeding manifestation.[5]

However, while essential thrombocythaemia may cause primary intracerebral haemorrhage (commonly if platelet counts exceed 1500 $\times 10^9$/L), secondary haemorrhage is of course a complication of intracranial venous thrombosis.

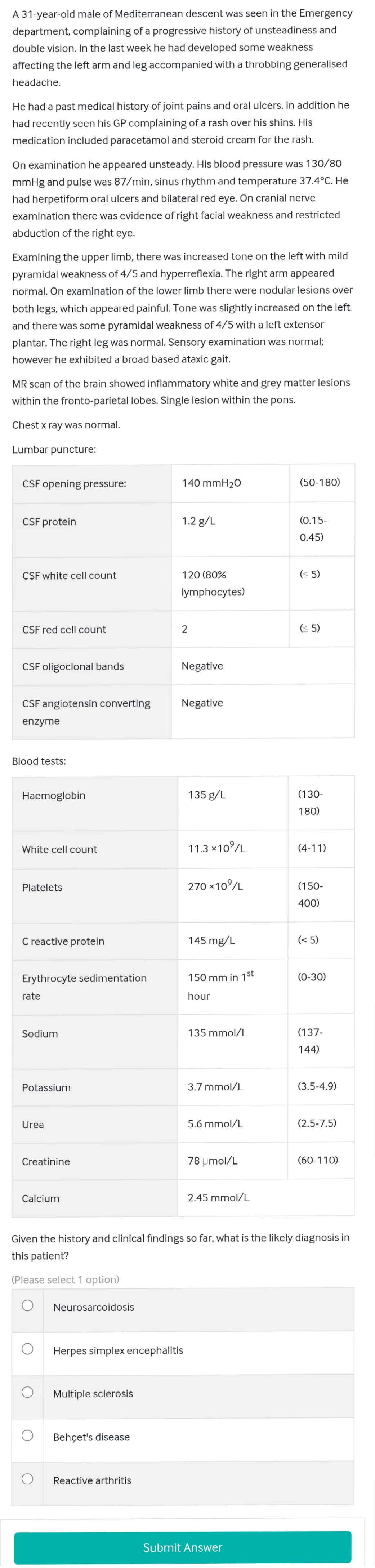
Clearly this is an important distinction as coming to the wrong diagnostic conclusion might adversely influence treatment - for example, inappropriate withdrawal of antithrombotics in patients with haemorrhagic intracranial venous thrombosis.[1]

The most appropriate course of action in regard to anticoagulation in this scenario is intravenous unfractionated heparin, with at least a doubled activated partial thromboplastin (a target activated partial thromboplastin time ratio of 1.5-2.5) with later transition to warfarin.[6]

Low dose aspirin and hydroxycarbamide is part of the treatment for ET in high risk patients (over 60 years, previous thrombotic episode and platelet count >1500 $\times 10^9$/L)[7]. This alone will not be sufficient to treat CVST in this scenario.

The combination of plasma exchange and corticosteroids is a mainstain of treatment in TTP (thrombotic thrombocytopenic purpura) but not in essential thrombocytosis. This patient will additionally need cytoreductive therapy with hydroxycarbamide but acute treatment of CVST must be instituted.

There is currently no evidence from randomised controlled trials about the efficacy and safety of either systemic or local thrombolytic therapy in patients with CVST. [6]



A 31-year-old male of Mediterranean descent was seen in the Emergency department, complaining of a progressive history of unsteadiness and double vision. In the last week he had developed some weakness affecting the left arm and leg accompanied with a throbbing generalised headache.

He had a past medical history of joint pains and oral ulcers. In addition he had recently seen his GP complaining of a rash over his shins. His medication included paracetamol and steroid cream for the rash.

On examination he appeared unsteady. His blood pressure was 130/80 mmHg and pulse was 87/min, sinus rhythm and temperature 37.4°C. He had herpetiform oral ulcers and bilateral red eye. On cranial nerve examination there was evidence of right facial weakness and restricted abduction of the right eye.

Examining the upper limb, there was increased tone on the left with mild pyramidal weakness of 4/5 and hyperreflexia. The right arm appeared normal. On examination of the lower limb there were nodular lesions over both legs, which appeared painful. Tone was slightly increased on the left and there was some pyramidal weakness of 4/5 with a left extensor plantar. The right leg was normal. Sensory examination was normal; however he exhibited a broad based ataxic gait.

MR scan of the brain showed inflammatory white and grey matter lesions within the fronto-parietal lobes. Single lesion within the pons.

Chest x ray was normal.

Lumbar puncture:

CSF opening pressure:	140 mmH_2O	(50-180)
CSF protein	1.2 g/L	(0.15-0.45)
CSF white cell count	120 (80% lymphocytes)	(≤ 5)
CSF red cell count	2	(≤ 5)
CSF oligoclonal bands	Negative	
CSF angiotensin converting enzyme	Negative	

Blood tests:

Haemoglobin	135 g/L	(130-180)
White cell count	11.3 $\times 10^9$/L	(4-11)
Platelets	270 $\times 10^9$/L	(150-400)
C reactive protein	145 mg/L	(< 5)
Erythrocyte sedimentation rate	150 mm in 1st hour	(0-30)
Sodium	135 mmol/L	(137-144)
Potassium	3.7 mmol/L	(3.5-4.9)
Urea	5.6 mmol/L	(2.5-7.5)
Creatinine	78 μmol/L	(60-110)
Calcium	2.45 mmol/L	

Given the history and clinical findings so far, what is the likely diagnosis in this patient?

(Please select 1 option)

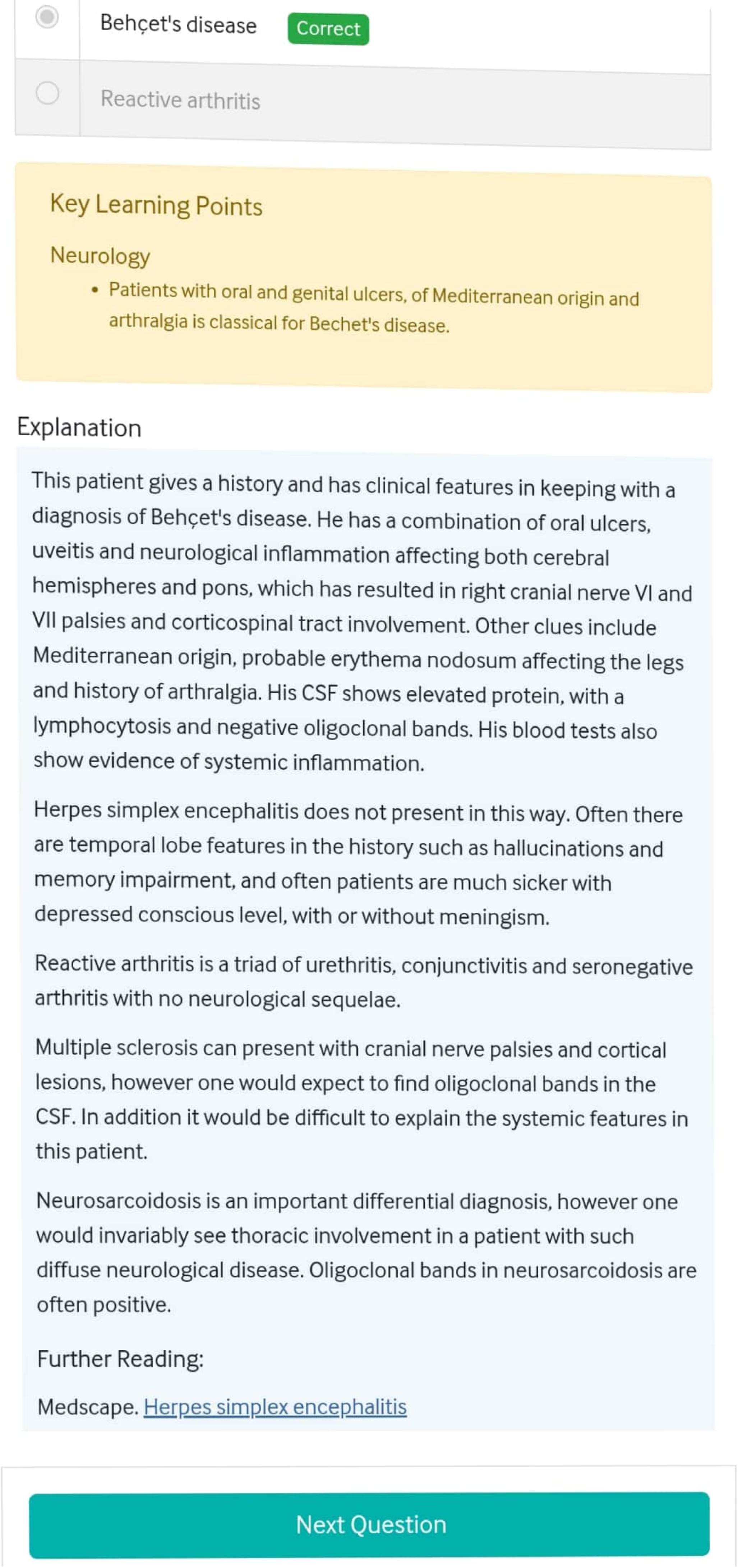
○ Neurosarcoidosis

○ Herpes simplex encephalitis

○ Multiple sclerosis

○ Behçet's disease

○ Reactive arthritis



Key Learning Points

Neurology

- Patients with oral and genital ulcers, of Mediterranean origin and arthralgia is classical for Bechet's disease.

Explanation

This patient gives a history and has clinical features in keeping with a diagnosis of Behçet's disease. He has a combination of oral ulcers, uveitis and neurological inflammation affecting both cerebral hemispheres and pons, which has resulted in right cranial nerve VI and VII palsies and corticospinal tract involvement. Other clues include Mediterranean origin, probable erythema nodosum affecting the legs and history of arthralgia. His CSF shows elevated protein, with a lymphocytosis and negative oligoclonal bands. His blood tests also show evidence of systemic inflammation.

Herpes simplex encephalitis does not present in this way. Often there are temporal lobe features in the history such as hallucinations and memory impairment, and often patients are much sicker with depressed conscious level, with or without meningism.

Reactive arthritis is a triad of urethritis, conjunctivitis and seronegative arthritis with no neurological sequelae.

Multiple sclerosis can present with cranial nerve palsies and cortical lesions, however one would expect to find oligoclonal bands in the CSF. In addition it would be difficult to explain the systemic features in this patient.

Neurosarcoidosis is an important differential diagnosis, however one would invariably see thoracic involvement in a patient with such diffuse neurological disease. Oligoclonal bands in neurosarcoidosis are often positive.

Further Reading:

Medscape. Herpes simplex encephalitis

CamScanner الممسوحة ضوئيا بـ

The wife of a 48-year-old man with Parkinson's disease has contacted you for some advice as she has found out that her husband has been spending huge sums of money gambling. Her recent credit card statement alerted her to the increased spending and she has found that he is gambling excessively online. He used to gamble regularly and this is not a new thing for him, but the amount of money he is now spending is vastly greater than anything he used to spend gambling. His wife also says that he has never kept his gambling secret from her, whereas now he seems to be concealing his actions.

He was diagnosed with Parkinson's disease at a young age about 8 years ago. She tells you that he currently seems quite stable in terms of the motor symptoms of his Parkinson's disease and is not acutely unwell. His mood can be a little bit variable but this is nothing new as he has always been frustrated by his diagnosis and the physical symptoms of the disease. His sleep is OK and he is eating well.

He is prescribed levodopa and carbidopa as a combination treatment (co-careldopa) and ropinirole.

Which of the following is the most likely cause of this problem?

(Please select 1 option)

○ Bipolar disorder

○ Iatrogenic

○ Acute delirium

○ Psychosis

○ Depression

Submit Answer

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● Iatrogenic Correct

○ Acute delirium

○ Psychosis

○ Depression

Key Learning Points

Neurology

- Impulse control disorder is an uncommon disorder in those using dopaminergic medication. Features include: hypersexuality, pathological gambling, and binge eating.

Explanation

Impulsive and compulsive behaviour is an uncommon but important possible side effect of some anti-Parkinsonian medication.

Parkinson's UK have a useful information pack on this issue which is worth a read.

Patients started on levodopa or a dopamine agonist should be warned of this potential problem so that they are their family/carers can monitor for any suspicious behaviour.

Further reading:

Impulsive and compulsive behaviour in Parkinson's

Next Question

A 58-year-old woman was referred to a neurologist having developed worsening headaches particularly in the morning. She described the headaches as throbbing and generalised associated with nausea. She felt they were positional and appeared to worsen on lying down.

She had recently noticed problems with her sense of smell and had developed difficulties seeing out of her right eye. She did not complain of any pain or diplopia but did mention that she had been bumping into things particularly on her left side.

She had hypertension, hypercholesterolaemia and ischaemic heart disease and took regular aspirin, ramipril and simvastatin. She was a smoker of 10 cigarettes per day and drank four units of alcohol per week.

On examination her blood pressure was 145/80 mmHg, pulse was 67/min and regular and heart sounds were normal. She had bilateral non-tender pulsatile temporal arteries.

Cranial nerve examination revealed sluggish direct pupillary reflex on the right with a relative afferent pupillary defect but normal on the left. Fundoscopy revealed right optic atrophy and mild swelling of the left optic disc with peripapillary haemorrhages. Visual field testing revealed a central scotoma on the right and restriction of visual fields on the left. Ocular movements were full and the rest of the cranial nerves were normal. Examination of the peripheral nervous system did not reveal any abnormalities.

Investigations revealed:

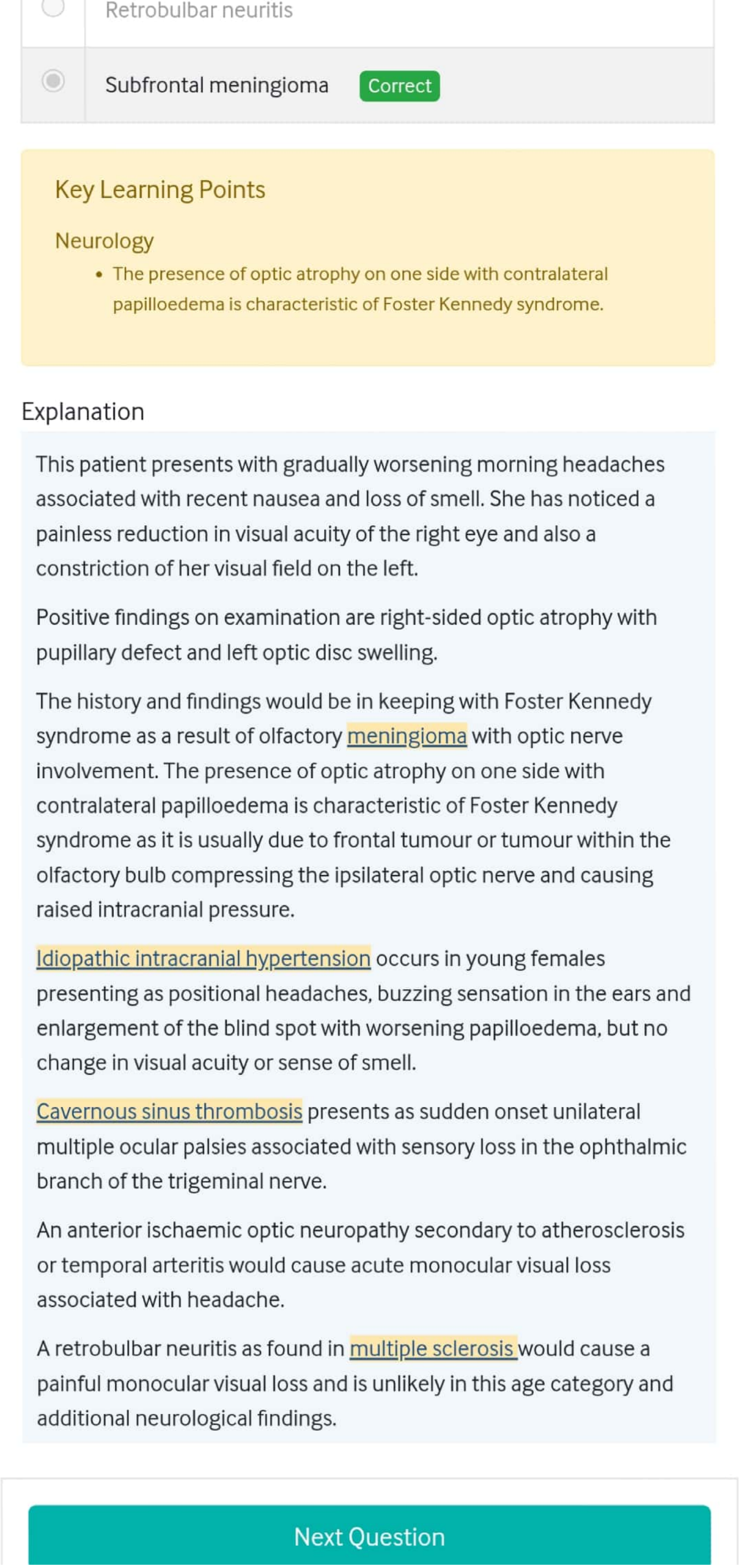
Haemoglobin	139 g/L	(130-180)
White cell count	9.3 $\times 10^9$/L	(4-11)
Platelets	207 $\times 10^9$/L	(150-400)
ESR (Westergren)	10 mm/1st hour	(0-15)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	4.1 mmol/L	(3.5-4.9)
Serum urea	3.2 mmol/L	(2.5-7.5)
Serum creatinine	99 µmol/L	(60-110)

What is the most likely cause for this patient's symptoms?

(Please select 1 option)

○	Cavernous sinus thrombosis
○	Idiopathic intracranial hypertension
○	Anterior ischaemic optic neuropathy
○	Retrobulbar neuritis
○	Subfrontal meningioma

Submit Answer



○ Retrobulbar neuritis

● Subfrontal meningioma **Correct**

Key Learning Points

Neurology

- The presence of optic atrophy on one side with contralateral papilloedema is characteristic of Foster Kennedy syndrome.

Explanation

This patient presents with gradually worsening morning headaches associated with recent nausea and loss of smell. She has noticed a painless reduction in visual acuity of the right eye and also a constriction of her visual field on the left.

Positive findings on examination are right-sided optic atrophy with pupillary defect and left optic disc swelling.

The history and findings would be in keeping with Foster Kennedy syndrome as a result of olfactory meningioma with optic nerve involvement. The presence of optic atrophy on one side with contralateral papilloedema is characteristic of Foster Kennedy syndrome as it is usually due to frontal tumour or tumour within the olfactory bulb compressing the ipsilateral optic nerve and causing raised intracranial pressure.

Idiopathic intracranial hypertension occurs in young females presenting as positional headaches, buzzing sensation in the ears and enlargement of the blind spot with worsening papilloedema, but no change in visual acuity or sense of smell.

Cavernous sinus thrombosis presents as sudden onset unilateral multiple ocular palsies associated with sensory loss in the ophthalmic branch of the trigeminal nerve.

An anterior ischaemic optic neuropathy secondary to atherosclerosis or temporal arteritis would cause acute monocular visual loss associated with headache.

A retrobulbar neuritis as found in multiple sclerosis would cause a painful monocular visual loss and is unlikely in this age category and additional neurological findings.

A 20-year-old female student presents with sleepiness, weakness and vivid dreams which have occurred over the last two months.

She has a six year history of type 1 diabetes and has been using basal bolus insulin consisting of three times daily Lyspro insulin and evening long acting insulin for the last six months. Prior to that she had been receiving twice daily insulin but had noted rather erratic control after she attended university.

She generally adhered to a good diet and regularly monitored her BMs twice daily which tend to be below 10. She is receiving no other treatment except insulin. She does not smoke, lives in a flat with two other female student colleagues and binge drinks often on Saturday nights.

Examination reveals a well female with a BMI of 23 kg/m^2, a pulse of 80 bpm and a blood pressure of 112/70 mmHg. No abnormalities are noted on examination.

Investigations reveal:

Haemoglobin	152 g/L	(115-165)
White cell count	6.8 ×10^9/L	(4-11)
Platelets	280 ×10^9/L	(150-400)
Serum sodium	146 mmol/L	(137-144)
Serum potassium	3.9 mmol/L	(3.5-4.9)
Serum urea	5.5 mmol/L	(2.5-7.5)
Serum creatinine	88 µmol/L	(60-110)
Plasma glucose	7.9 mmol/L	(3.0-6.0)
HbA$_{1c}$	6.2%	(<6%)

What is the most likely explanation for her symptoms?

(Please select 1 option)

○ Temporal lobe epilepsy

○ Addison's disease

○ Narcolepsy syndrome

○ Hypoglycaemic episodes

○ Sleep apnoea

Submit Answer

CamScanner المسوحة ضوئيا بـ

○ Narcolepsy syndrome

● Hypoglycaemic episodes Correct

○ Sleep apnoea

Key Learning Points

Diabetes, Endocrinology, Neurology

- Vivid dreams and lethargy may be due to nocturnal hypoglycaemia

Explanation

Seizures at night are associated with sleep disruption and deprivation of REM sleep, and are thus not generally associated with vivid dreams.

Narcolepsy is associated with hypersomnolence, cataplexy, sleep paralysis and hypnagogic hallucinations and is not classically associated with vivid dreams.

Sleep apnoea is associated with vivid dreams, but would be very unlikely in a young, non-obese female.

Schizophrenia is an unlikely cause of this symptom.

The most likely cause of this symptom of vivid dreams in this patient is nocturnal hypoglycaemia.

REM sleep disruption may lead to daytime weakness and somnolence.

Addison's disease is unlikely given her BP and sodium

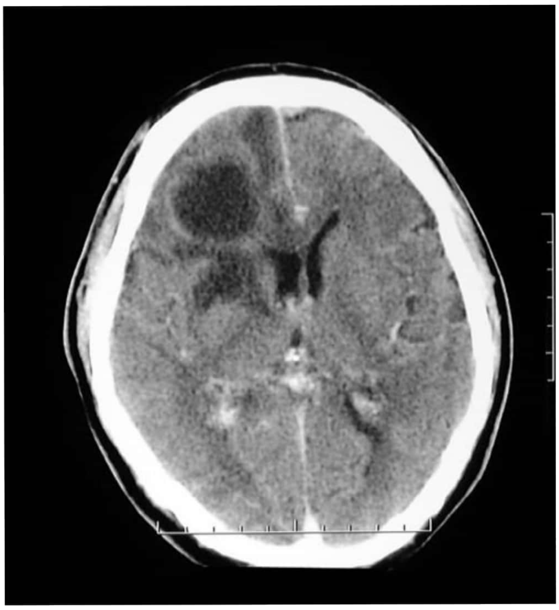
Next Question

A 20-year-old veterinary nurse was admitted to the emergency department with two generalised seizures. Her boyfriend, who had been there at the time, said she became vacant and began jerking her left arm. After several seconds she had collapsed to the floor jerking all limbs. She had bitten her tongue and had been incontinent of urine.

In the past few days she had been complaining of headaches and had recently attended her GP for sinusitis and had completed a course of oral penicillin. Otherwise she was fit and well and did not take any regular medication. She was a non-smoker and did not drink alcohol.

On examination her temperature was 39.2°C, blood pressure was 115/75 mmHg and pulse was 90 beats per minute. She appeared flushed and confused. There was some mild neck stiffness and generalised cervical lymphadenopathy. Cranial nerve examination did not reveal any obvious abnormalities and fundoscopy was normal. Examination of the peripheral nervous system revealed generalised hyperreflexia with equivocal plantar responses. There was slightly increased tone in the left arm, but the rest of the examination appeared normal. Chest and abdominal examinations were normal.

A CT scan of the brain was performed. An image from this study, after administration of intravenous contrast, is shown below:



A lumbar puncture was performed and yielded the following data:

Opening pressure	17 cm H_2O	(6-18 cm)
CSF white cell count	120 cells per mL	(<5)
CSF white cell differential	90% lymphocytes	
CSF red cell count	4 cells per mL	(<5)
CSF protein	0.75 g/L	0.15-0.45)
CSF glucose	3.6mmol/L	(3.3-4.4)
Cytospin	Negative for cells	

The chest x ray was normal.

What is the most likely diagnosis in this patient?

(Please select 1 option)

○ Pyogenic brain abscess

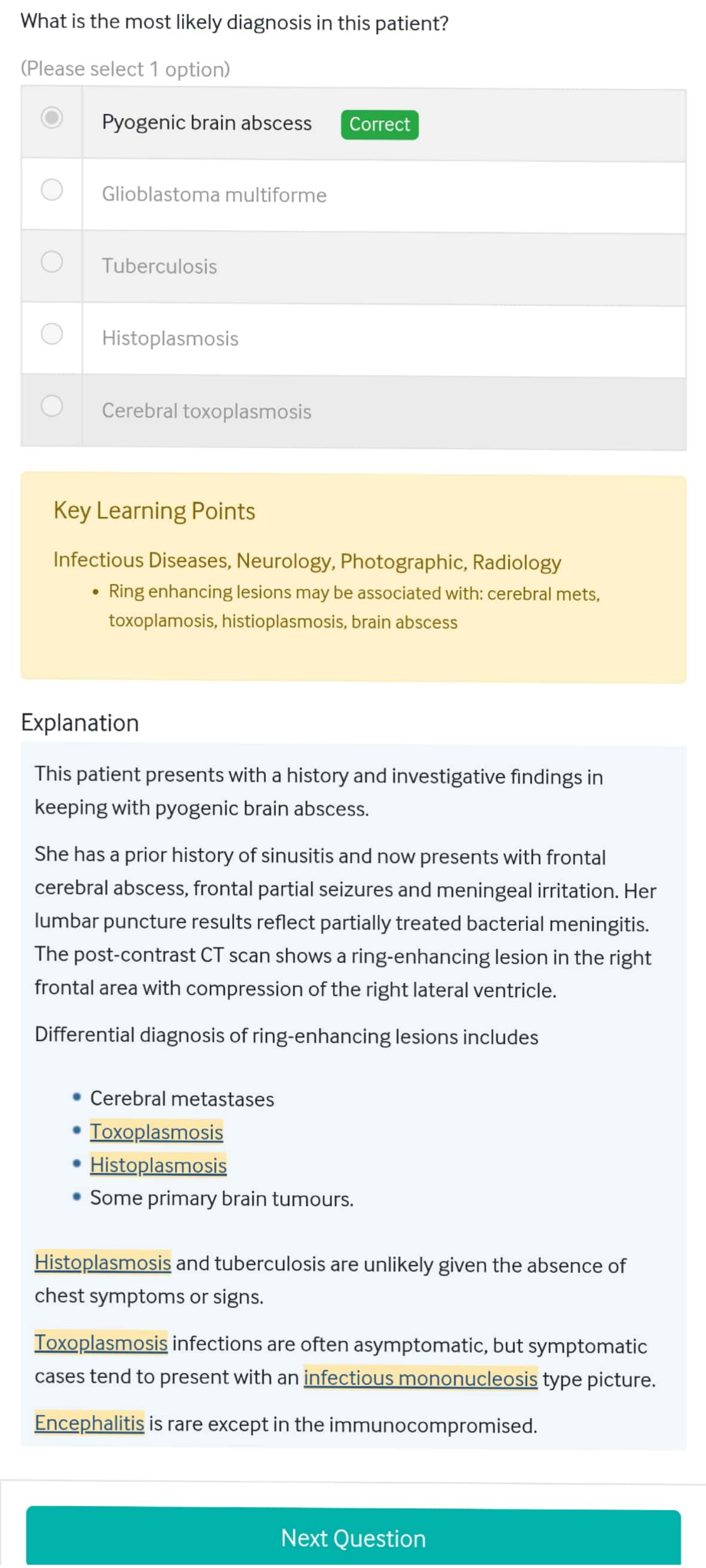
○ Glioblastoma multiforme

○ Tuberculosis

○ Histoplasmosis

○ Cerebral toxoplasmosis

Submit Answer



What is the most likely diagnosis in this patient?

(Please select 1 option)

● Pyogenic brain abscess **Correct**

○ Glioblastoma multiforme

○ Tuberculosis

○ Histoplasmosis

○ Cerebral toxoplasmosis

Key Learning Points

Infectious Diseases, Neurology, Photographic, Radiology

- Ring enhancing lesions may be associated with: cerebral mets, toxoplamosis, histioplasmosis, brain abscess

Explanation

This patient presents with a history and investigative findings in keeping with pyogenic brain abscess.

She has a prior history of sinusitis and now presents with frontal cerebral abscess, frontal partial seizures and meningeal irritation. Her lumbar puncture results reflect partially treated bacterial meningitis. The post-contrast CT scan shows a ring-enhancing lesion in the right frontal area with compression of the right lateral ventricle.

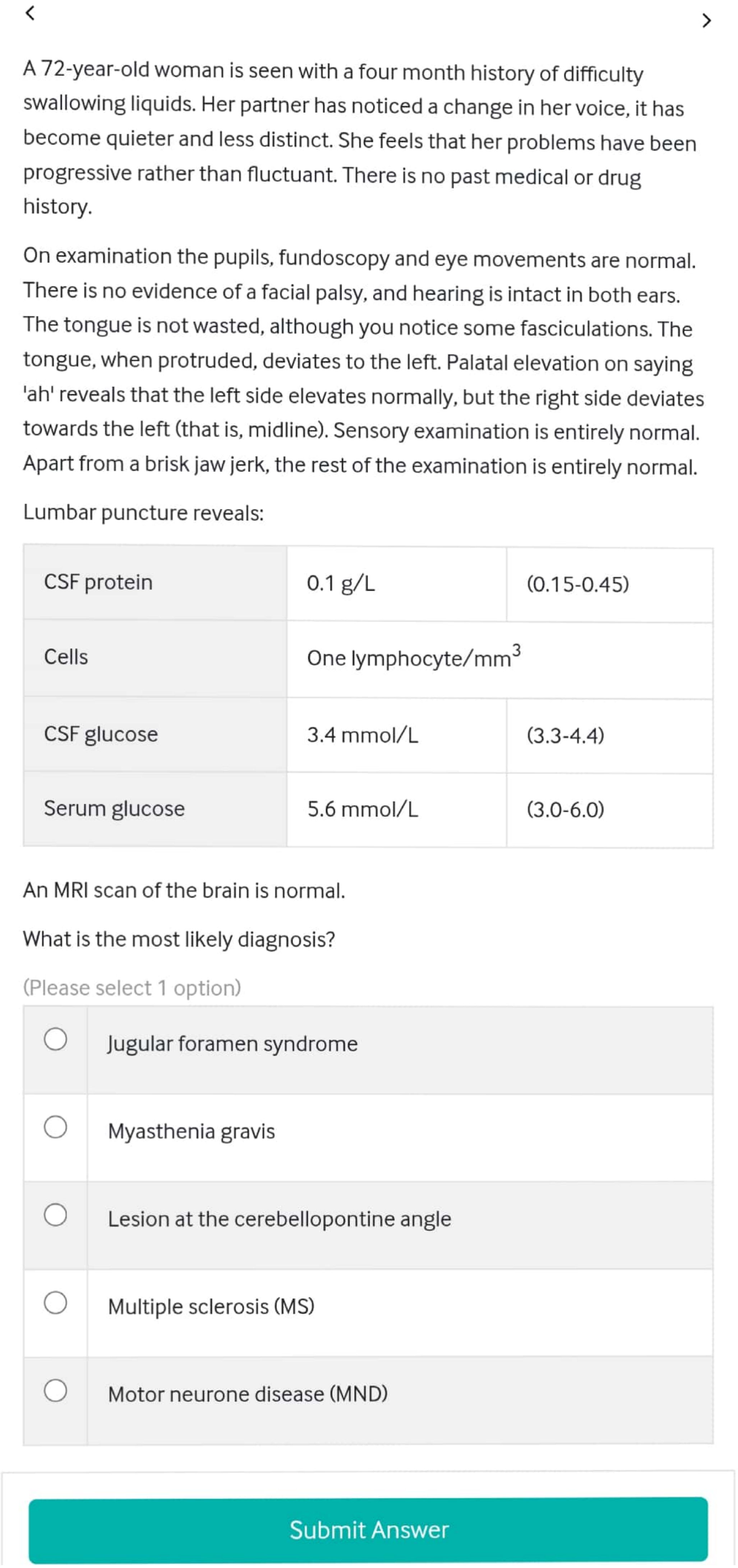
Differential diagnosis of ring-enhancing lesions includes

- Cerebral metastases
- Toxoplasmosis
- Histoplasmosis
- Some primary brain tumours.

Histoplasmosis and tuberculosis are unlikely given the absence of chest symptoms or signs.

Toxoplasmosis infections are often asymptomatic, but symptomatic cases tend to present with an infectious mononucleosis type picture.

Encephalitis is rare except in the immunocompromised.



A 72-year-old woman is seen with a four month history of difficulty swallowing liquids. Her partner has noticed a change in her voice, it has become quieter and less distinct. She feels that her problems have been progressive rather than fluctuant. There is no past medical or drug history.

On examination the pupils, fundoscopy and eye movements are normal. There is no evidence of a facial palsy, and hearing is intact in both ears. The tongue is not wasted, although you notice some fasciculations. The tongue, when protruded, deviates to the left. Palatal elevation on saying 'ah' reveals that the left side elevates normally, but the right side deviates towards the left (that is, midline). Sensory examination is entirely normal. Apart from a brisk jaw jerk, the rest of the examination is entirely normal.

Lumbar puncture reveals:

CSF protein	0.1 g/L	(0.15-0.45)
Cells	One lymphocyte/mm^3	
CSF glucose	3.4 mmol/L	(3.3-4.4)
Serum glucose	5.6 mmol/L	(3.0-6.0)

An MRI scan of the brain is normal.

What is the most likely diagnosis?

(Please select 1 option)

○ Jugular foramen syndrome

○ Myasthenia gravis

○ Lesion at the cerebellopontine angle

○ Multiple sclerosis (MS)

○ Motor neurone disease (MND)

○ Multiple sclerosis (MS)

○ Motor neurone disease (MND) **This is the correct answer**

Key Learning Points

Neurology

- The combination of upper and lower motor signs with the absence of sensory involvement is suggestive of MND.

Explanation

This is essentially the differential diagnosis of a bulbar palsy.

The main feature of the presentation is a bulbar palsy, although brisk jaw jerk is unusual in this setting.

Myasthenia is unlikely in the absence of fluctuating course and eye signs.

Multiple sclerosis is also unlikely, as this illness is monophasic with a pattern of distribution which would be unusual for MS. The normal MRI supports this assertion.

It is unlikely that a lesion at the cerebellopontine angle or jugular foramen would be missed by MRI. The pattern does not suggest a CP angle lesion (VII and VIII are intact), a jugular foramen syndrome would also tend to affect the shoulder and neck.

The signs are bilateral (left XII, right IX/X). The combination of upper and lower motor signs with the absence of sensory involvement makes MND the probable diagnosis.

Next Question

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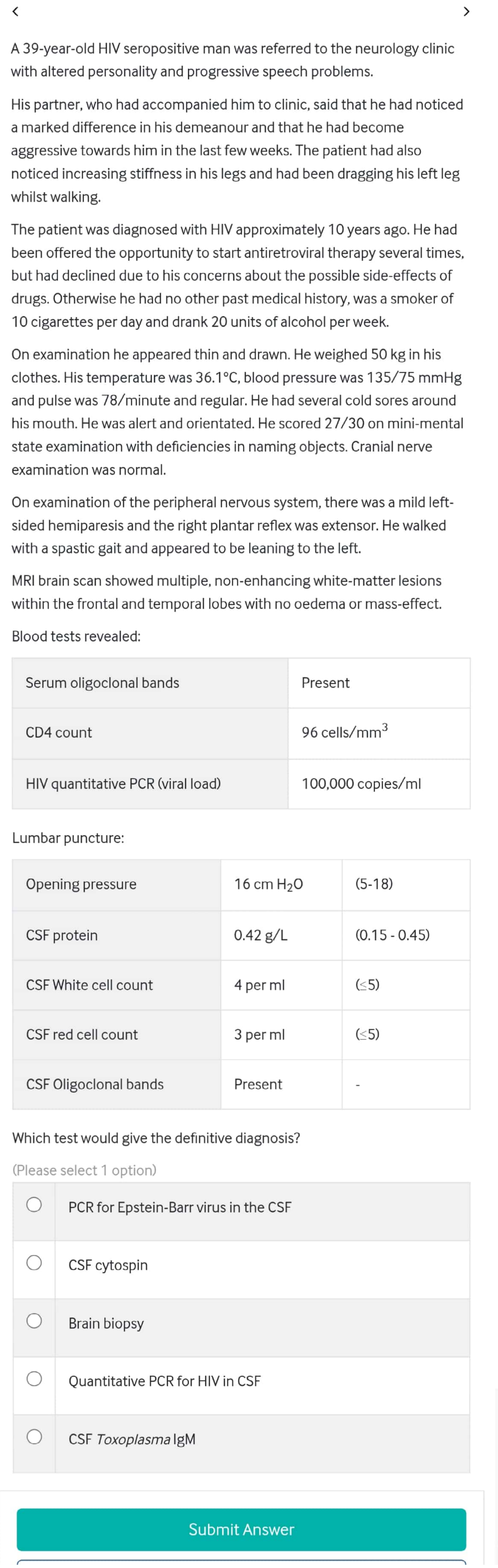

A 39-year-old HIV seropositive man was referred to the neurology clinic with altered personality and progressive speech problems.

His partner, who had accompanied him to clinic, said that he had noticed a marked difference in his demeanour and that he had become aggressive towards him in the last few weeks. The patient had also noticed increasing stiffness in his legs and had been dragging his left leg whilst walking.

The patient was diagnosed with HIV approximately 10 years ago. He had been offered the opportunity to start antiretroviral therapy several times, but had declined due to his concerns about the possible side-effects of drugs. Otherwise he had no other past medical history, was a smoker of 10 cigarettes per day and drank 20 units of alcohol per week.

On examination he appeared thin and drawn. He weighed 50 kg in his clothes. His temperature was 36.1°C, blood pressure was 135/75 mmHg and pulse was 78/minute and regular. He had several cold sores around his mouth. He was alert and orientated. He scored 27/30 on mini-mental state examination with deficiencies in naming objects. Cranial nerve examination was normal.

On examination of the peripheral nervous system, there was a mild left-sided hemiparesis and the right plantar reflex was extensor. He walked with a spastic gait and appeared to be leaning to the left.

MRI brain scan showed multiple, non-enhancing white-matter lesions within the frontal and temporal lobes with no oedema or mass-effect.

Blood tests revealed:

Serum oligoclonal bands	Present
CD4 count	96 cells/mm^3
HIV quantitative PCR (viral load)	100,000 copies/ml

Lumbar puncture:

Opening pressure	16 cm H_2O	(5-18)
CSF protein	0.42 g/L	(0.15 - 0.45)
CSF White cell count	4 per ml	(≤5)
CSF red cell count	3 per ml	(≤5)
CSF Oligoclonal bands	Present	-

Which test would give the definitive diagnosis?

(Please select 1 option)

○ PCR for Epstein-Barr virus in the CSF

○ CSF cytospin

○ Brain biopsy

○ Quantitative PCR for HIV in CSF

○ CSF *Toxoplasma* IgM

○ Brain biopsy **This is the correct answer**

○ Quantitative PCR for HIV in CSF

○ CSF *Toxoplasma* IgM

Key Learning Points

Neurology

- A brain biopsy is the definitive diagnostic test for PML.

Explanation

This patient presents with a left hemiparesis, expressive dysphasia and behavioural changes in clear consciousness. There is absence of fever or depressed conscious level, which would be more in keeping with lymphoma, HIV encephalopathy or abscess.

The diagnosis is likely to be progressive multifocal leukoencephalopathy.

Progressive multifocal leukoencephalopathy is the result of infection of oligodendrocytes with JC virus, which is a papovavirus found latent in most healthy adults.

With CD4 counts of less than 100 this virus becomes active leading to progressive neurological deterioration. Hemiparesis, visual field defects, ataxia and mental change are characteristic.

A brain biopsy would be the definitive diagnostic test showing asymmetric foci of demyelination and intranuclear inclusions containing the JC virus.

There is no effective treatment, but progression can be slowed by initiation of antiretroviral therapy.

CamScanner الممسوحة ضوئيا بـ

A 69-year-old man presented to his GP with a four-month history of weakness of his lower limbs. He had noticed some difficulty walking up and down stairs and more recently had noticed a constant dry mouth and mild drooping of his eyelids. He denied any problems with swallowing or any visual disturbances and had not noticed any change in sensation in his legs, or alteration in bladder function.

His past medical history included a recent chest infection for which he had taken a course of antibiotics. He was a smoker of 20 cigarettes per day and did not take any regular medication. On examination he appeared alert and orientated. There appeared to be generalised reduced muscle bulk but no fasciculations. His blood pressure was 139/78mmHg, pulse was 67/min and regular and temperature was 36.7°C. He had mild bilateral ptosis however ocular movements were full and there was no apparent neck weakness or swallowing impairment.

On peripheral nervous system examination the were no obvious abnormalities in the upper limb, however on lower limb examination there was marked weakness of hip flexion and extension, symmetrically reduced reflexes and no demonstrable sensory disturbances. On chest examination he had bilateral expiratory wheeze with reduced air entry and dull percussion note in the right lung base.

Blood tests show:

Haemoglobin	176 g/L	(130 - 180)
White cell count	11.3 $\times 10^9$/L	(4 - 11)
Platelets	478 $\times 10^9$/L	(150 - 400)
Serum sodium	130 mmol/L	(137 - 144)
Serum potassium	3.5 mmol/L	(3.5 - 4.9)
Serum creatinine	104 µmol/L	(60 - 110)
Serum urea	4.4 mmol/L	(2.5 - 7.5)
Serum creatine kinase	103 U/L	(24 - 195)
ESR (Westergren)	<5 in 1st hour	(0 - 20)

What is the most likely diagnosis?

(Please select 1 option)

○ Guillain-Barré syndrome

○ Lambert-Eaton Syndrome

○ Myasthenia gravis

○ Polymyositis

○ Motor neuron disease

● Lambert-Eaton Syndrome **Correct**

○ Myasthenia gravis

○ Polymyositis

○ Motor neuron disease

Key Learning Points

Neurology

- Lambert-Eaton syndrome is a paraneoplastic disorder usually associated with small cell lung carcinoma

Explanation

This patient gives a history of proximal lower limb weakness, facial weakness and autonomic dysfunction in absence of sensory, bulbar or respiratory compromise. There is a history of smoking and the suggestion of a right-sided pleural effusion on respiratory examination. The most likely diagnosis is Lambert-Eaton syndrome associated with lung malignancy.

This is a paraneoplastic disorder usually associated with small cell lung carcinoma. It is a presynaptic neuromuscular disorder associated with decreased release of acetylcholine from the presynaptic cleft. It is associated with positive voltage-gated calcium channel antibodies.

Clinical features include:

- autonomic symptoms
- proximal lower limb weakness
- reduced reflexes, and
- mild ptosis.

Another possibility would be polymyositis, which is associated with malignancy. However positive tensilon and normal creatine kinase would go against this diagnosis.

This is not a history for Guillain-Barré syndrome, which would present more acutely usually with a history of preceding infection and sensory disturbance or radicular pain.

Motor neurone disease presents with a combination of upper and lower motor neurone signs

Next Question

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A 55-year-old lady is brought to the Emergency department at 7 pm by her son who claims that she has become confused and is acting strangely. The patient claims to be completely asymptomatic and does not understand why she was brought to this place.

On further questioning the patient claims to have arrived home at 3 pm and gone to sleep and then her son brought her here thereafter. Her son however describes that he found his mother roaming around the house and could not recognise his wife, thinking she was some new girlfriend of his. He is very worried about the fact that he had to remind her continuously they were at the hospital.

The patient does not have any medical or surgical history of note. She was previously well until this afternoon when she has had a normal working day as a sales person in a bookshop.

On examination there are no neurological deficits and fundoscopy is normal. Of note, the patient is co-operative but looks surprisingly calm and unconcerned about her condition.

What is the best line of management for this condition?

(Please select 1 option)

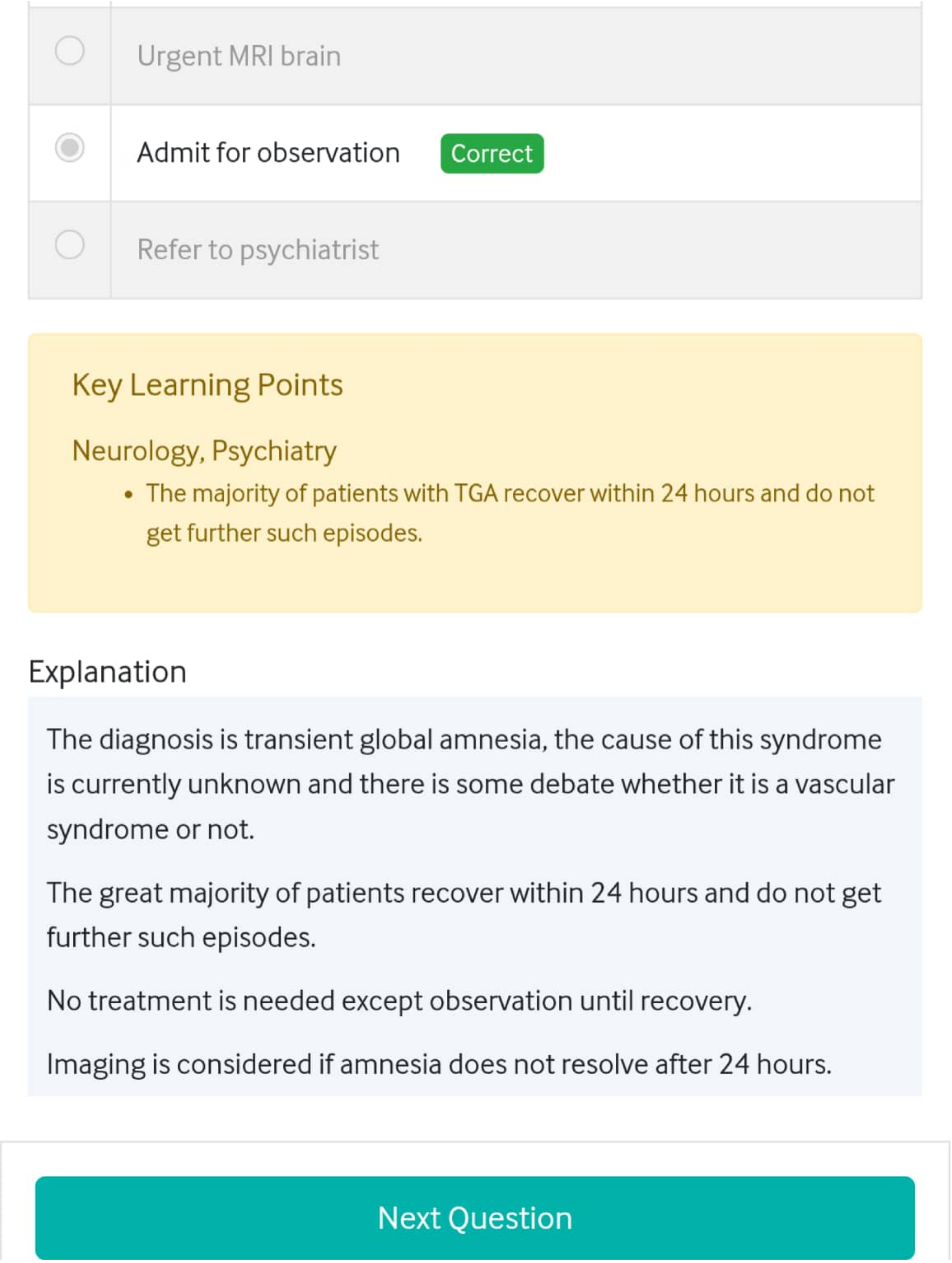
○ Start haloperidol

○ Urgent CT scan brain

○ Urgent MRI brain

○ Admit for observation

○ Refer to psychiatrist



○ Urgent MRI brain

● Admit for observation **Correct**

○ Refer to psychiatrist

Key Learning Points

Neurology, Psychiatry

- The majority of patients with TGA recover within 24 hours and do not get further such episodes.

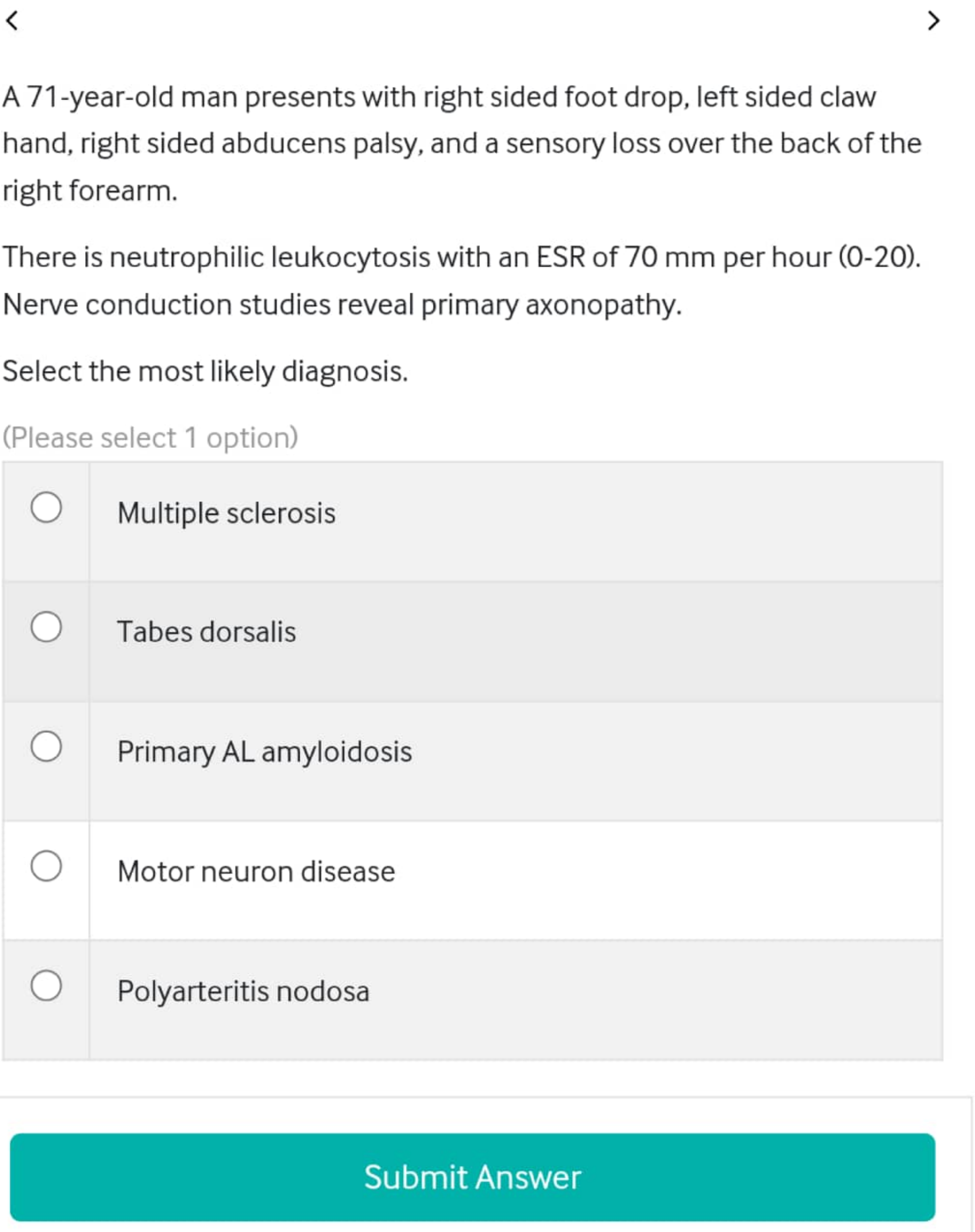
Explanation

The diagnosis is transient global amnesia, the cause of this syndrome is currently unknown and there is some debate whether it is a vascular syndrome or not.

The great majority of patients recover within 24 hours and do not get further such episodes.

No treatment is needed except observation until recovery.

Imaging is considered if amnesia does not resolve after 24 hours.



A 71-year-old man presents with right sided foot drop, left sided claw hand, right sided abducens palsy, and a sensory loss over the back of the right forearm.

There is neutrophilic leukocytosis with an ESR of 70 mm per hour (0-20). Nerve conduction studies reveal primary axonopathy.

Select the most likely diagnosis.

(Please select 1 option)

- ○ Multiple sclerosis
- ○ Tabes dorsalis
- ○ Primary AL amyloidosis
- ○ Motor neuron disease
- ○ Polyarteritis nodosa

○ Multiple sclerosis

○ Tabes dorsalis

○ Primary AL amyloidosis

○ Motor neuron disease

● Polyarteritis nodosa **Correct**

Key Learning Points

Neurology

- Primary axonopathy is seen in vasculitis.

Explanation

This man with mononeuritis multiplex (note the distribution) and neutrophilic leukocytosis, high erythrocyte sedimentation rate and primary axonopathy, is likely to have a "vasculitis", so polyarteritis nodosa and granulomatosis with polyangiitis should be the top differential diagnoses.

Subacute primary axonopathy should prompt a search for vasculitic, toxic, and metabolic causes of neuropathy.

Next Question

A 30-year-old woman presented to hospital after returning from a holiday in France.

She had had a cold three weeks prior to the holiday, but during the holiday had woken up with severe pain around the right shoulder. On returning home she developed some proximal weakness of the right arm, despite the pain subsiding somewhat. She had no significant past medical history.

She was married, lived with her husband and they did not have any children. She was a smoker of 10 cigarettes per day and drank 10 units of alcohol per week. She and her husband were vintage car enthusiasts who restored classic cars. They had driven to France in one of their restored cars, a vintage Daimler, and had been participating in car rallies and motor racing events. She denied any injuries through motor racing and did not have any known problems with her neck.

On examination of the upper limb there was full range of passive movement at the right shoulder joint with some mild proximal muscle wasting. Tone appeared slightly reduced at the elbow and wrist and the biceps and supinator jerk were both depressed. On examination of power, there was 3/5 power of right shoulder abduction and elbow flexion with mild weakness of elbow extension. Sensation was reduced to all modalities over the deltoid. There were no cerebellar signs in the upper limb. Lower limb examination was entirely normal. Nerve conduction studies showed multifocal denervation in the deltoid and biceps.

Lumbar puncture showed:

Opening pressure	140 mmH_2O	(50-180)
CSF protein	0.56 g/L	(0.15-0.45)
CSF white cell count	5 cells per ml	($\leq$5)
CSF red cell count	4 cells per ml	($\leq$5)

What is the most likely diagnosis?

(Please select 1 option)

○ Multifocal motor neuropathy

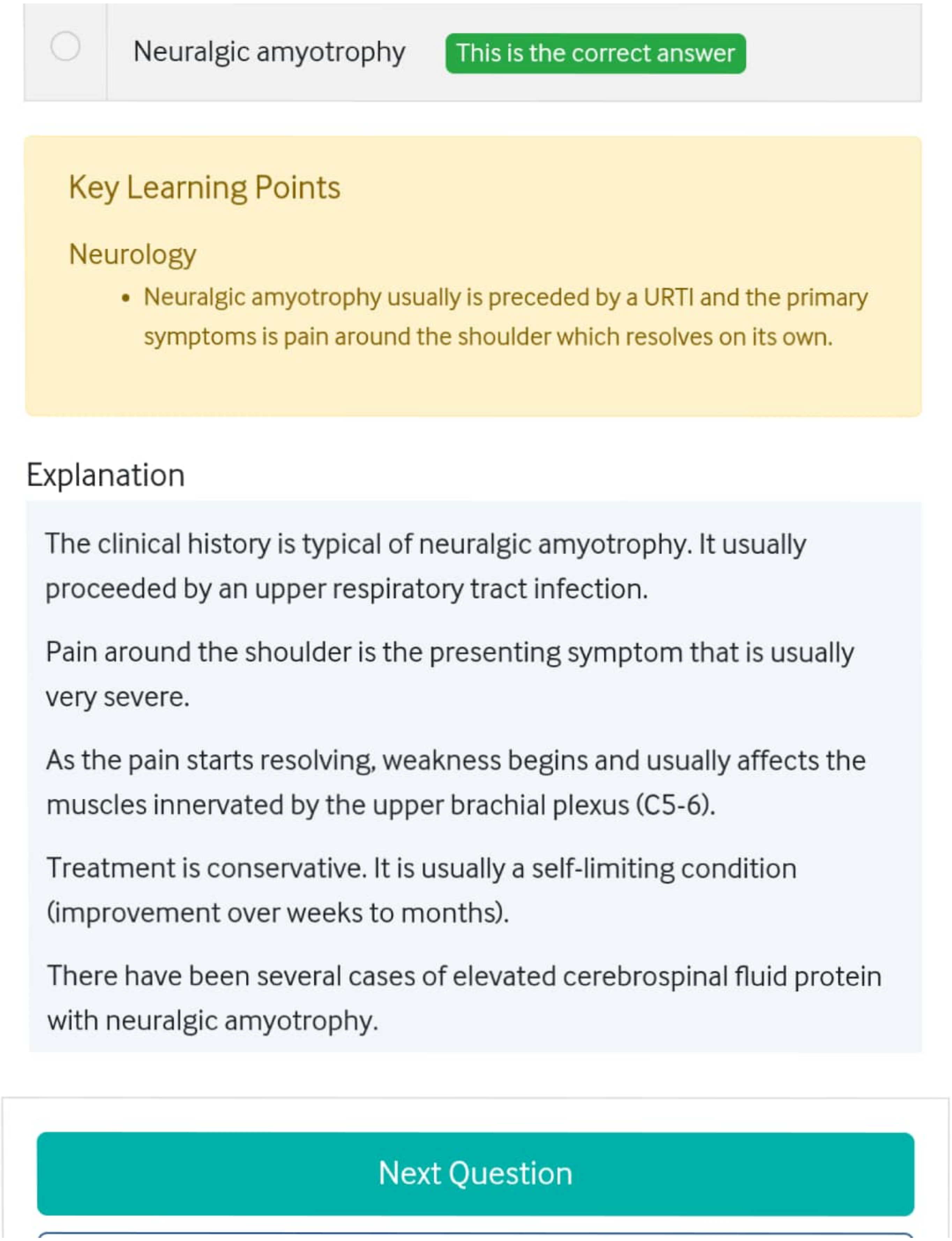
○ Erb's palsy

○ Circumflex nerve injury

○ C5 disc prolapse

○ Neuralgic amyotrophy

Submit Answer



○ Neuralgic amyotrophy

Key Learning Points

Neurology

- Neuralgic amyotrophy usually is preceded by a URTI and the primary symptoms is pain around the shoulder which resolves on its own.

Explanation

The clinical history is typical of neuralgic amyotrophy. It usually proceeded by an upper respiratory tract infection.

Pain around the shoulder is the presenting symptom that is usually very severe.

As the pain starts resolving, weakness begins and usually affects the muscles innervated by the upper brachial plexus (C5-6).

Treatment is conservative. It is usually a self-limiting condition (improvement over weeks to months).

There have been several cases of elevated cerebrospinal fluid protein with neuralgic amyotrophy.

A 22-year-old man with a history of ataxia, hand tremor and muscle spasms affecting his arms, legs and face was referred by his GP to a neurologist.

On occasions the spasms have been so sustained that it has led to abnormal posturing of his legs and arms. His friends have noticed that he was walking as if drunk. More recently he had complained of joint pains.

He had no past medical history and had normal development and achievement of milestones. He was not on any regular medication; however there was a family history of early onset dementia.

On general examination he had a yellow tinge to the skin and small spider naevi over the chest wall. In addition there were small bruises over both arms.

Chest and cardiovascular examination was normal and abdominal examination appeared normal. He had sunflower cataracts on fundoscopy, cranial nerve examination was normal however, he had some facial grimacing.

Examining the upper limb, he had a bilateral intention tremor and experienced some spasms of the left arm with dystonic posturing. Tone, power and reflexes were all normal. On examination of the lower limb he had some dystonic posturing of the right leg, with normal power tone and reflexes. He exhibited a broad-based ataxic gait.

MR brain scan showed high T2 signal in the basal ganglia, thalami, dentate nuclei, and cerebellar white matter.

Blood tests revealed:

Haemoglobin	115 g/L	(130-180)
White cell count	7.8 ×10^9/L	(4-11)
Platelets	254 ×10^9/L	(150-400)
Blood film	Normal	-
Sodium	134 mmol/L	(137-144)
Potassium	3.5 mmol/L	(3.5-4.9)
Urea	7.8 mmol/L	(2.5-7.5)
Creatinine	129 µmol/L	(60-110)
Bilirubin	35 µmol/L	(1-22)
Alanine aminotransferase	79 U/L	(5-35)
Aspartate transaminase	88 U/L	(1-31)
Alkaline phosphatase	105 U/L	(45-105)
Albumin	24 g/L	(37-49)
Prothrombin time	20.4 seconds	(11.5-15.5)
Activated partial thromboplastin time	53 seconds	(30-40)
Calcium	2.00 mmol/L	(2.2-2.6)
Phosphate	1.46 mmol/L	(0.8-1.40)

Given the history and investigations so far, what further test would you arrange to make the diagnosis?

(Please select 1 option)

- ○ Copper studies
- ○ Liver biopsy
- ○ Homocysteine levels
- ○ Slit lamp investigations
- ○ Autoimmune screen

Submit Answer

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(Please select 1 option)

- ● Copper studies **Correct**
- ○ Liver biopsy
- ○ Homocysteine levels
- ○ Slit lamp investigations
- ○ Autoimmune screen

Key Learning Points

Neurology

- Copper studies in Wilson's disease show low serum copper, low serum caeruloplasmin and increased urinary copper.

Explanation

This patient has a history and clinical findings in keeping with Wilson disease. This is characterised by copper accumulation in the liver, brain and kidney as a result of defective ATPases involved in the excretion of copper into bile.

Copper accumulation in the liver leads to hepatitis and progression to liver cirrhosis with stigmata of chronic liver disease.

The basal ganglia and cerebellum are mainly affected by copper accumulation leading to movement disorders (parkinsonism, chorea and dystonia) and cerebellar syndrome.

The gold standard diagnostic test is a liver biopsy; however in this patient he has significantly deranged clotting which would be hazardous.

Kayser-Fleischer rings found by slit lamp analysis are found in the majority of patients with neurological sequelae. In some patients however, they can be absent.

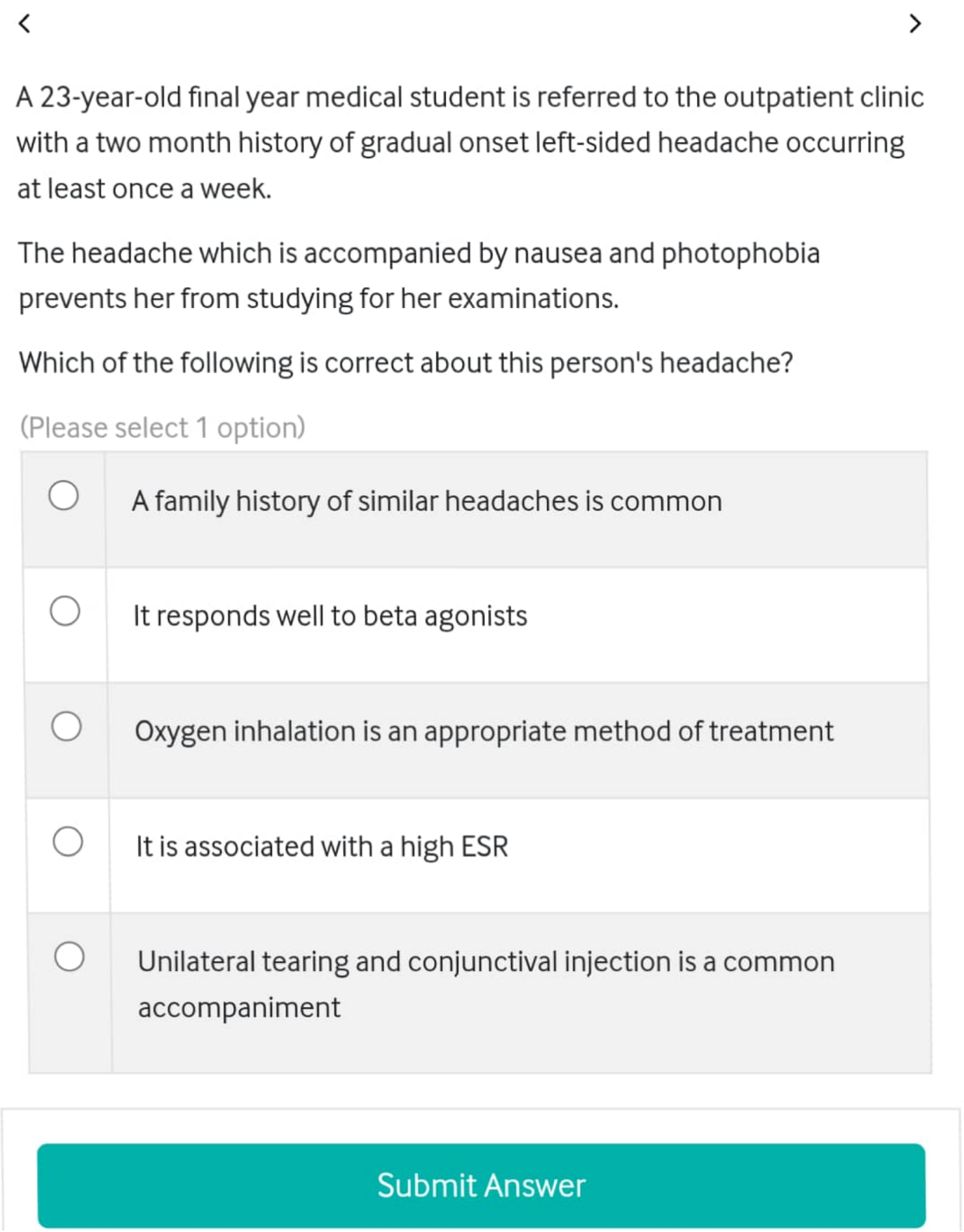
Copper studies should be carried out in this patient looking for low serum copper level (normal 80-160 mcg/dL), low serum caeruloplasmin level, less than 20 mcg/dL (normal 20-60 mcg/dL) and increased urinary copper level more than 100 mcg/24 h (normal 10-80 mcg/24 h).

Other notable features in this patient are evidence of osteomalacia, chronic liver disease and sunflower cataracts, all associated with Wilson disease.

Homocysteinuria as a result of homocysteine accumulation in tissues results in thromboembolic neurological complications and psychiatric disturbances.

Next Question

CamScanner الممسوحة ضوئيا بـ



A 23-year-old final year medical student is referred to the outpatient clinic with a two month history of gradual onset left-sided headache occurring at least once a week.

The headache which is accompanied by nausea and photophobia prevents her from studying for her examinations.

Which of the following is correct about this person's headache?

(Please select 1 option)

○	A family history of similar headaches is common
○	It responds well to beta agonists
○	Oxygen inhalation is an appropriate method of treatment
○	It is associated with a high ESR
○	Unilateral tearing and conjunctival injection is a common accompaniment

prevents her from studying for her examinations.

Which of the following is correct about this person's headache?

(Please select 1 option)

- ● A family history of similar headaches is common **Correct**
- ○ It responds well to beta agonists
- ○ Oxygen inhalation is an appropriate method of treatment
- ○ It is associated with a high ESR
- ○ Unilateral tearing and conjunctival injection is a common accompaniment

Key Learning Points

Neurology

- Migraine is more common where there is a family history of it.

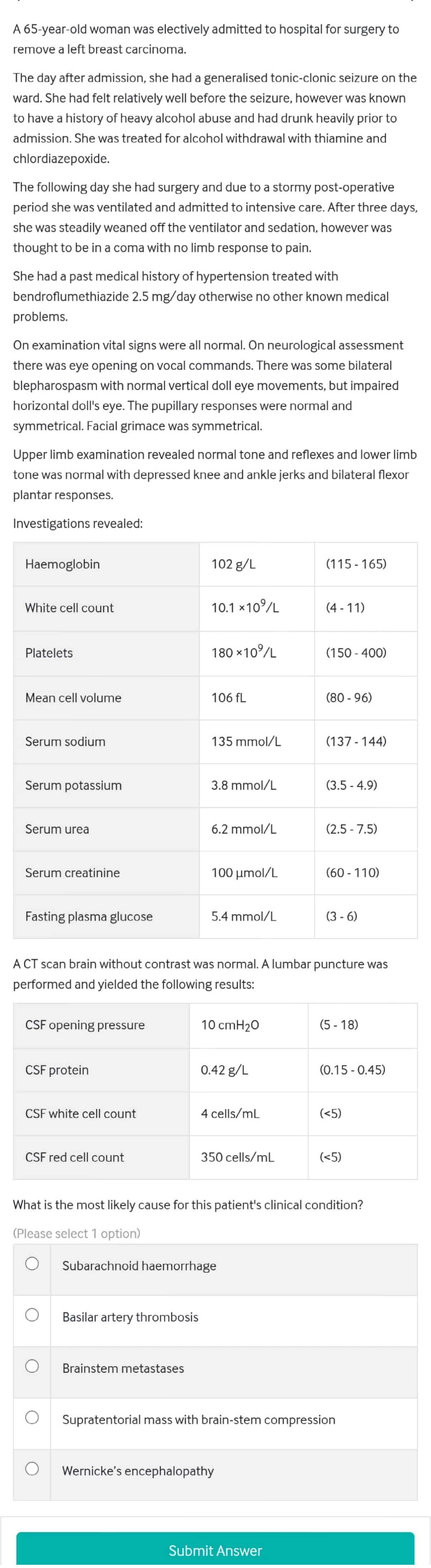
Explanation

In most cases where a family history is present the headache is a migraine. It will respond to beta blockers.

Tearing and conjunctival injection are features of cluster headache for which acute relief may be achieved by inhaling 100% oxygen for 10-15 minutes.

Temporal arteritis which is associated with a high erythrocyte sedimentation rate is unusual before the age of 60 years.

Next Question



A 65-year-old woman was electively admitted to hospital for surgery to remove a left breast carcinoma.

The day after admission, she had a generalised tonic-clonic seizure on the ward. She had felt relatively well before the seizure, however was known to have a history of heavy alcohol abuse and had drunk heavily prior to admission. She was treated for alcohol withdrawal with thiamine and chlordiazepoxide.

The following day she had surgery and due to a stormy post-operative period she was ventilated and admitted to intensive care. After three days, she was steadily weaned off the ventilator and sedation, however was thought to be in a coma with no limb response to pain.

She had a past medical history of hypertension treated with bendroflumethiazide 2.5 mg/day otherwise no other known medical problems.

On examination vital signs were all normal. On neurological assessment there was eye opening on vocal commands. There was some bilateral blepharospasm with normal vertical doll eye movements, but impaired horizontal doll's eye. The pupillary responses were normal and symmetrical. Facial grimace was symmetrical.

Upper limb examination revealed normal tone and reflexes and lower limb tone was normal with depressed knee and ankle jerks and bilateral flexor plantar responses.

Investigations revealed:

Haemoglobin	102 g/L	(115 - 165)
White cell count	10.1 $\times 10^9$/L	(4 - 11)
Platelets	180 $\times 10^9$/L	(150 - 400)
Mean cell volume	106 fL	(80 - 96)
Serum sodium	135 mmol/L	(137 - 144)
Serum potassium	3.8 mmol/L	(3.5 - 4.9)
Serum urea	6.2 mmol/L	(2.5 - 7.5)
Serum creatinine	100 μmol/L	(60 - 110)
Fasting plasma glucose	5.4 mmol/L	(3 - 6)

A CT scan brain without contrast was normal. A lumbar puncture was performed and yielded the following results:

CSF opening pressure	10 cmH_2O	(5 - 18)
CSF protein	0.42 g/L	(0.15 - 0.45)
CSF white cell count	4 cells/mL	(<5)
CSF red cell count	350 cells/mL	(<5)

What is the most likely cause for this patient's clinical condition?

(Please select 1 option)

○ Subarachnoid haemorrhage

○ Basilar artery thrombosis

○ Brainstem metastases

○ Supratentorial mass with brain-stem compression

○ Wernicke's encephalopathy

CamScanner المسوحة ضوئيا بـ

○ Supratentorial mass with brain-stem compression

○ Wernicke’s encephalopathy **This is the correct answer**

Key Learning Points

Neurology

- In Wernicke's encephalopthy patients should receive IV thiamine and B12.

Explanation

This patient has impaired horizontal eye movements with preserved pupillary responses and absence of any upper motor neurone focal signs peripherally.

The unenhanced CT scan is normal suggesting integrity of the brainstem and the unlikelihood of subarachnoid haemorrhage, which is 90% sensitive if performed within 24 hours.

The lumbar puncture is normal other than a raised red cell count, nevertheless, the sample should be sent for spectophotometry.

The most likely cause is 'metabolic coma'. We are given the history of alcohol abuse, and there is a macrocytosis.

Wernicke's encephalopathy is most likely which can result in abnormal horizontal eye movements, ataxia, confusion or present as coma. Thiamine should be continued in this patient.

Brainstem metastases or basilar artery thrombosis would lead to pupillary abnormalities and corticospinal disruption.

A supratentorial mass with brain-stem compression would lead to unilateral focal neurology and pupillary abnormalities namely third nerve compression.

A 48-year-old woman presented to the Emergency department having developed gradual weakness initially affecting the left arm and more recently involving weakness affecting the left leg. She complained of severe intractable headaches associated with recent photophobia. Her daughter who accompanied her said that recently she appeared to be muddled and had forgotten several appointments in the last week.

She had no previous medical history and did not take any regular medication. She was a non-smoker and did not drink any alcohol. There was no family history of any medical problems and both her daughters were in good health.

On examination she appeared confused. Her blood pressure was 138/78 mmHg, pulse was 78/minute and regular and temperature was 36.4°C. Heart sounds were normal and chest was clear. Fundoscopy examination revealed mild bilateral papilloedema. Pupillary responses were normal and ocular movements were full. There were no other abnormalities detected in the rest of the cranial nerves.

Examination of the upper limb revealed pyramidal weakness of the left arm associated with hyperreflexia and hypertonia. Sensation appeared intact. Lower limb examination revealed pyramidal weakness with brisk reflexes and a left extensor plantar response. There also appeared to be some mild right-sided cerebellar signs of pastpointing and heel shin ataxia. The patient went on to have several investigations outlined below.

Blood tests:

Full blood count	normal
Erythrocyte sedimentation rate	10 mm/1st hour
ANCA antibodies	negative
Biochemistry	normal

A lumbar puncture was performed and yielded the following data:

Opening pressure	12 cm H_2O	(5 - 18)
CSF protein	0.78 g/L	(0.15 - 0.45)
CSF white cell count	120 cells per ml	(<5)
CSF white cell differential	90% lymphocytes	-
CSF red cell count	4 cells per ml	(<5)
CSF lactate	1.5 mmol/L	(1 - 2)
CSF oligoclonal bands	Negative	-
CSF culture	Negative	-

A MRI scan of the brain showed multiple irregular deep white matter lesions.

What is the likely diagnosis?

(Please select 1 option)

- ○ Binswanger's disease
- ○ CADASIL
- ○ Multiple sclerosis
- ○ MELAS
- ○ Cerebral angiitis

Submit Answer

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○ MELAS

○ Cerebral angiitis **This is the correct answer**

Key Learning Points

Neurology

- Progressive multifocal neurology involving cerebral cortex, cerebellum and meninges may be a sing of cerebral angiitis.

Explanation

This patient presents with progressive multifocal neurology involving cerebral cortex, cerebellum and meninges. She has signs and symptoms in keeping with aseptic meningitis, left-sided pyramidal dysfunction and right-sided cerebellar dysfunction. She gives a history of intractable headaches in absence of a history of migraine or family history of migraine. The most likely diagnosis in this patient would be cerebral angiitis.

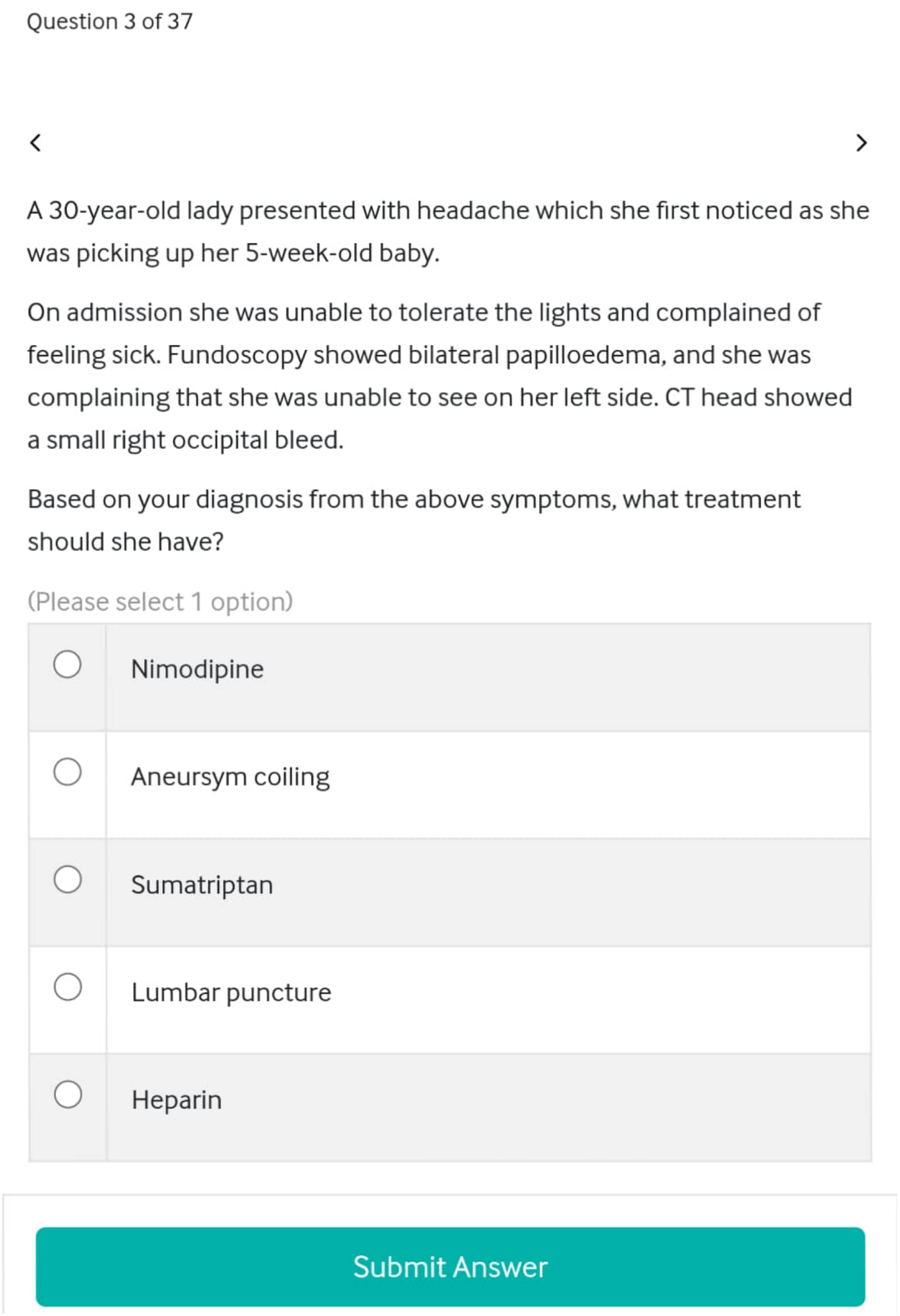
Inflammatory markers are often normal in primary cerebral angiitis, and diagnosis is only confirmed in 50% of cases using cerebral angiography. Some patients require cerebral biopsy to clinch the diagnosis. Treatment is with immunosuppression including steroids and cyclophosphamide.

In absence of hypertension, Binswanger's disease is unlikely.

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is associated with migraine, recurrent lacunar strokes and dementia.

Mitochondrial disorders are unlikely given the normal cerebrospinal fluid lactate, and multiple sclerosis is unlikely in this age group and in absence of intrathecal oligoclonal bands.

Next Question



Question 3 of 37

A 30-year-old lady presented with headache which she first noticed as she was picking up her 5-week-old baby.

On admission she was unable to tolerate the lights and complained of feeling sick. Fundoscopy showed bilateral papilloedema, and she was complaining that she was unable to see on her left side. CT head showed a small right occipital bleed.

Based on your diagnosis from the above symptoms, what treatment should she have?

(Please select 1 option)

- ○ Nimodipine
- ○ Aneursym coiling
- ○ Sumatriptan
- ○ Lumbar puncture
- ○ Heparin

○ Lumbar puncture

○ Heparin **This is the correct answer**

Key Learning Points

Neurology

- In Venous sinus thrombopsis anticoagilation should be continued even if there is evidence of bleeding intracranially.

Explanation

This lady has developed a venous sinus thrombosis peri-partum resulting in symptoms of headache, photophobia and vomiting. The small occipital bleed has resulted from venous congestion and she needs urgent treatment with low molecular weight heparin (LWMH) to prevent clot propagation and further complications.

This woman's symptoms are not as a result of a subarachnoid aneurysm, so aneurysm coiling and nimodipine are not appropriate.

A lumbar puncture is not appropriate as she does not have idiopathic intracranial hypertension.

She does not have migraine and so a triptan is not appropriate.

Next Question

A 26-year-old woman was admitted to the Emergency department complaining of diplopia and headaches.

She had started with the headache in the morning, which she described as generalised and throbbing in nature associated with some nausea. In the last two hours she had noticed pain and diplopia on looking to the right, and her left eyelid had started to droop. She had not noticed any change in facial sensation, or any weakness in her arms or legs.

She had a past medical history of migraine and took occasional Imigran for persistent headaches. Otherwise she was fit and well. She was a smoker of seven cigarettes per day and drank 16 units of alcohol per week.

On examination she was alert and orientated. Her blood pressure was 132/78 mmHg with no postural drop and pulse was 78/minute and regular. There was some mild nuchal rigidity. Fundoscopy did not reveal any abnormalities but there was evidence of photophobia.

Cranial nerve examination revealed a left oculomotor nerve palsy with left-sided ptosis and mydriasis. There was no evidence of chemosis or proptosis. The rest of the cranial nerves and visual fields appeared normal. There were no abnormalities detected in the peripheral nervous system.

Investigations revealed:

Haemoglobin	134 g/L	(115-165)
White cell count	6.2 $\times 10^9$/L	(4-11)
Platelets	199 $\times 10^9$/L	(150-400)
Serum sodium	139 mmol/L	(137-144)
Serum potassium	3.1 mmol/L	(3.5-4.9)
Serum urea	4.9 mmol/L	(2.5-7.5)
Serum creatinine	101 µmol/L	(60-110)
C reactive protein	10 mg/L	(<10)

Given the clinical account, which investigation is likely to clinch the diagnosis?

(Please select 1 option)

- ○ Trial of sumatriptan
- ○ Pituitary function tests
- ○ MR venogram
- ○ MR brain scan
- ○ Intra-arterial digital subtraction angiography

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○ Intra-arterial digital subtraction angiography

This is the correct answer

Key Learning Points

Neurology

- Digital subtraction angiography may be helpful in identifying an acutely ruptured aneurysm in SAH.

Explanation

The differential diagnoses in a patient presenting with headaches and painful diplopia are:

- A posterior communicating aneurysm (PCA)
- Ophthalmoplegic migraine
- Pituitary adenoma
- Cavernous sinus thrombosis, or
- A medical mononeuritis.

This patient has features suggestive of a posterior communicating artery aneurysm. She has:

- Pupillary dilatation
- Ophthalmoplegia, and
- Ptosis.

In addition she has other features suggestive of subarachnoid blood (headache, nuchal rigidity and photophobia).

Conventional angiography is the definitive procedure for the detection and characterisation of cerebral aneurysms. Digital subtraction angiography may be helpful in identifying an acutely ruptured aneurysm.

Features distinguishing PCA from a cavernous sinus thrombosis are absence of sinusitis or a midface infection, which are common, absence of fever or additional cranial nerve abnormalities.

Although a pituitary adenoma or apoplexy can cause ophthalmoplegia, visual field defects or signs suggestive of pituitary dysfunction would be present.

Ophthalmoplegic migraine can present with episodic third nerve palsy and headache. However there should be suspicion in the absence of a migraine history, if headache is severe and persistent with total ophthalmoplegia, if the onset is after age 20 and if there are symptoms and signs of subarachnoid haemorrhage.

Next Question

You are referred an 18-year-old female with restricted eye movements and progressive muscle weakness.

At the age of 6 years she was noted to have a squint and was given glasses. At 7 she complained of double vision that gradually resolved. She did reasonably well until her early teens when she found herself unable to compete with other children at play. For the last one year her double vision has recurred and she has noticed progressive generalised weakness, for example difficulty rising from a seated to standing position.

On examination she was of short stature with ptosis and a paucity of spontaneous facial expressions. Mental status examination was normal. Eye movements were absent in all directions. The pupils responded to light. The visual fields were full to confrontation but her visual acuity was reduced even with correction. The fundi showed pigmentary degeneration. Cataracts were not present. Hearing was normal.

Motor examination revealed weakness of neck muscles and proximal muscle groups in the lower extremities. Deep tendon reflexes were reduced and plantar reflexes were flexor. Cerebellar testing revealed intact finger to nose, slow rapid alternating movements in the upper extremities with mild ataxia, moderate heel to shin ataxia and gait ataxia. Romberg's testing was steady with eyes open and closed. Sensory examination showed preserved sensation to all primary modalities. The rest of the systemic examination was unremarkable. ECG showed incomplete heart block.

Which of the following is not a complication of the patient's underlying diagnosis?

(Please select 1 option)

○ Hypoparathyroidism

○ Cortical blindness

○ Lactic acidosis

○ Sensorineural deafness

○ Diabetes mellitus

Submit Answer

○ Cortical blindness **This is the correct answer**

○ Lactic acidosis

○ Sensorineural deafness

○ Diabetes mellitus

Key Learning Points

Neurology

- InKearns-Sayre syndrome most cases have large mitochondrial DNA mutations and red ragged fibres on muscle biopsy.

Explanation

Kearns-Sayre syndrome produces the classic triad of

- Progressive external ophthalmoplegia
- Pigmentary degeneration of the retina and
- Heart block.

Most cases have large mitochondrial DNA mutations.

Ragged red fibres are seen on muscle biopsy.

Although increased cerebrospinal fluid protein concentrations on lumbar puncture and white matter damage on MRI are seen, these changes are not specific to the syndrome.

Cortical blindness is not a complication of the condition.

Next Question

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A 19-year-old man was admitted to the Emergency department, having been found unconscious in the toilets in a nightclub.

There was no one accompanying him, but he had apparently been seen staggering around the club in an anxious state several hours before. He was found with an empty packet of olanzapine and half a packet of lithium tablets in his pocket.

On examination there was vomitus around the mouth and a strong smell of alcohol. His Glasgow coma scale was 8/15 (eye opening 1, verbal 2, motor 6), and pupils were dilated but reactive. His blood pressure was 160/89 mmHg, pulse 102/min and rectal temperature was 38.2°C.

There was no neck stiffness, rash, and fundoscopy was normal. There were no cranial nerve palsies and on examination of the peripheral nervous system he had generalised hypertonia, brisk reflexes bilaterally and equivocal plantar responses.

An urgent CT scan was entirely normal.

Lumbar puncture showed:

Opening pressure	13 cmH_2O	(5-18)
Protein	0.35 g/L	(0.15-0.45)
White cell count	5 cells per ml	(≤5)
Red cell count	20 cells per ml	(≤5)
Xanthochromia	No	

Blood tests showed:

Sodium	128 mmol/L	(137-144)
Potassium	4.9 mmol/L	(3.5-4.9)
Urea	20.4 mmol/L	(2.5-7.5)
Creatinine	240 µmol/L	(60-110)
Creatine kinase	4000 U/L	(24-170)
Glucose	4.8 mmol/L	(3.5-5.5)
Haemoglobin	135 g/L	(130-180)
White cell count	11.0 $\times 10^9$/L	(4-11)
Platelets	289 $\times 10^9$/L	(150-400)

Urinalysis showed myoglobinuria +++.

What is the likely cause for this patient's symptoms?

(Please select 1 option)

○ LSD ingestion

○ Lithium toxicity

○ MDMA (ecstasy) overdose

○ Acute alcohol intoxication

○ Neuroleptic malignant syndrome

Submit Answer

○ MDMA (ecstasy) overdose **This is the correct answer**

○ Acute alcohol intoxication

● Neuroleptic malignant syndrome **Incorrect answer selected**

Key Learning Points

Neurology

- Rhabdomyolysis and associated renal failure are signs of MDMA overdose.

Explanation

This patient's findings would fit with a diagnosis of ecstasy overdose.

Clues in the history are apparent ataxia and agitation prior to loss of consciousness (probable seizure) and sympathetic over activity as evidenced by hypertension, hyperpyrexia and tachycardia. He has also developed rhabdomyolysis and associated renal failure.

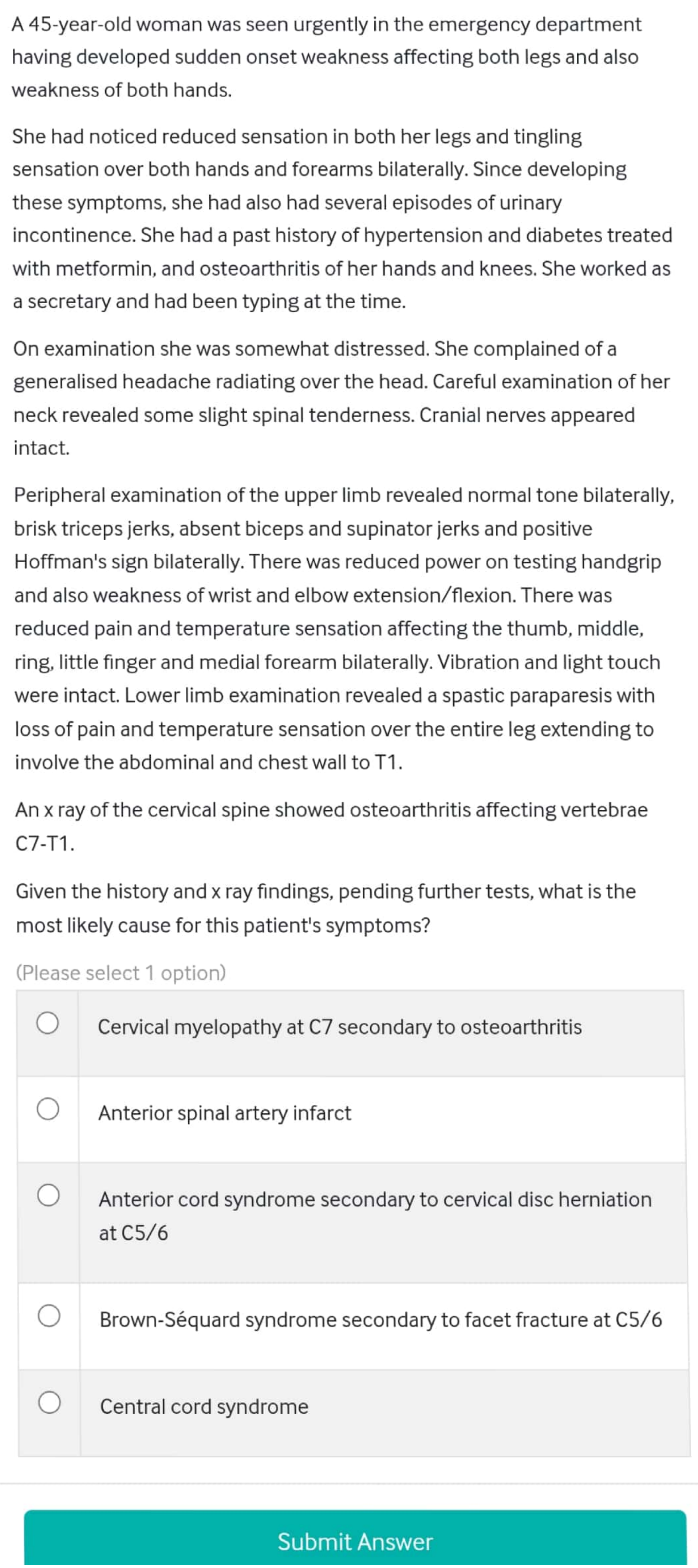
Alcohol intoxication has a sedative effect and would not result in sympathetic over activity.

Neuroleptic malignant syndrome associated with olanzapine ingestion results in extrapyramidal rigidity, and there is often a history of dysphagia, hypersalivation and incontinence.

Lithium toxicity can result in a cerebellar syndrome, but would not explain the additional sympathetic findings.

Lysergic acid diethylamide (LSD) has an effect on mood, perception and thought, but does not seem to be associated with any neurological problems.

Treatment for this patient includes aggressive rehydration, and cooling measures, control of seizures and monitoring of cardiac and renal function.



A 45-year-old woman was seen urgently in the emergency department having developed sudden onset weakness affecting both legs and also weakness of both hands.

She had noticed reduced sensation in both her legs and tingling sensation over both hands and forearms bilaterally. Since developing these symptoms, she had also had several episodes of urinary incontinence. She had a past history of hypertension and diabetes treated with metformin, and osteoarthritis of her hands and knees. She worked as a secretary and had been typing at the time.

On examination she was somewhat distressed. She complained of a generalised headache radiating over the head. Careful examination of her neck revealed some slight spinal tenderness. Cranial nerves appeared intact.

Peripheral examination of the upper limb revealed normal tone bilaterally, brisk triceps jerks, absent biceps and supinator jerks and positive Hoffman's sign bilaterally. There was reduced power on testing handgrip and also weakness of wrist and elbow extension/flexion. There was reduced pain and temperature sensation affecting the thumb, middle, ring, little finger and medial forearm bilaterally. Vibration and light touch were intact. Lower limb examination revealed a spastic paraparesis with loss of pain and temperature sensation over the entire leg extending to involve the abdominal and chest wall to T1.

An x ray of the cervical spine showed osteoarthritis affecting vertebrae C7-T1.

Given the history and x ray findings, pending further tests, what is the most likely cause for this patient's symptoms?

(Please select 1 option)

○ Cervical myelopathy at C7 secondary to osteoarthritis

○ Anterior spinal artery infarct

○ Anterior cord syndrome secondary to cervical disc herniation at C5/6

○ Brown-Séquard syndrome secondary to facet fracture at C5/6

○ Central cord syndrome

○ Anterior cord syndrome secondary to cervical disc herniation at C5/6 This is the correct answer

○ Brown-Séquard syndrome secondary to facet fracture at C5/6

○ Central cord syndrome

Key Learning Points

Neurology

- Anterior cord syndrome causes UMN and LMN signs acutely.

Explanation

This patient presents with symptoms and signs of an anterior cord syndrome, the cause of which is likely to be an acute disc herniation at the level of C5/C6.

In the lower limb there is upper motor neuron (UMN) weakness and evidence of spinothalamic disruption. In the upper limb there is lower motor neuron (LMN) signs at the level of C5/C6 as evidenced by absent biceps/supinator jerks and weak elbow flexion, and UMN signs below this level (brisk triceps jerk, positive Hoffman's sign, weakness of handgrip, wrist extension, elbow extension).

There also appears to be some evidence of spinothalamic disruption affecting C6-T1 bilaterally.

This patient needs urgent assessment with MRI imaging and referral to a neurosurgeon for decompression.

Anterior spinal artery infarct occurs at the 'watershed' T4-T6 and would cause symptoms primarily in the lower limb and a sensory level.

Brown-Séquard from a unilateral facet fracture causes ipsilateral loss of dorsal column and corticospinal function with contralateral loss of pain and temperature below the level of the lesion.

Central cord syndrome, that is, syringomyelia, would cause LMN weakness in the arms greater than UMN weakness in the legs, and dissociated pain and temperature loss in the arms.

A cervical myelopathy at C7 would not account for all the signs in the upper limb, and is unusual to have such a sudden onset.

Next Question

A 45-year-old woman was seen in the Emergency department having developed weakness and stiffness of her legs.

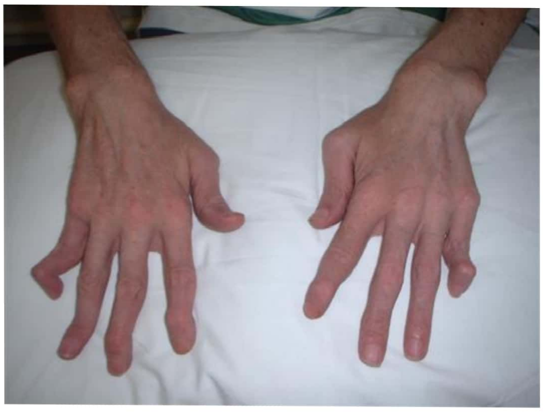
Her symptoms had been progressive over a number of months. She appeared to walk with a stiff gait and had had a number of falls. She had a history of joint pains affecting her hands and feet and had recently noticed pain in her back around the thoracic area. She had not noticed any change in her bowels, but had noticed increased urinary frequency.

She had attended her GP eight months ago with painful visual disturbance in her right eye, which seemed to recover after three days. No diagnosis was made at the time. Otherwise, she was fit and well, was a non-smoker and did not drink alcohol.

On examination she had multiple oral ulcers. Her blood pressure was 145/78 mmHg, pulse was 87 beats per minute and regular and she was apyrexial. Examination on the cranial nerves did not reveal any abnormalities and fundoscopy was entirely normal.

Upper limb examination revealed deformities of both hands as shown. Tone, power, reflexes and sensation in the upper limbs were intact. Lower limb examination revealed marked increase in tone with pyramidal weakness of 3/5 in both legs and pathologically brisk reflexes with bilateral extensor plantar responses. There was also reduced sensation to pinprick bilaterally to the level of T4.

A photograph of the patient's hands is shown below:



Investigations revealed:

- Rheumatoid factor negative
- Anti-nuclear antigen 1: 160
- Extractable nuclear antigen negative

A lumbar puncture was performed and yielded the following data:

Opening pressure	12 cm H_2O	(6 - 18)
CSF protein	0.79 g/L	(0.15 - 0.45)
CSF white cell count	10 cells per mL	(<5)
CSF white cell differential	90% lymphocytes	-
CSF red cell count	2 cells per mL	(<5)
CSF oligoclonal bands	Positive	-
Serum oligoclonal bands	Positive	-

A plain x ray of the hands did not reveal any bone deformity.

What is the likely diagnosis in this patient?

(Please select 1 option)

○ Myelopathy secondary to rheumatoid arthritis

○ Multiple sclerosis

○ Myelopathy secondary to osteoarthritis

○ Systemic lupus erythematosus

○ Reactive arthritis

Submit Answer

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○ Myelopathy secondary to osteoarthritis

● Systemic lupus erythematosus **Correct**

○ Reactive arthritis

Key Learning Points

Infectious Diseases, Neurology, Photographic, Rheumatology

- Jaccoud's arthropathy may be seen in SLE, RA, Parkinson's disease and hypocompementaemic urticarial vasculitis

Explanation

This patient presents with a subacute myelopathy in the presence of a positive anti-nuclear antigen, lymphocytic pleocytosis and oligoclonal bands.

In addition she also has oral ulcers and a Jaccoud's arthropathy. The picture shows joint subluxations and swan neck deformities, caused by recurrent episodes of synovitis that damage tendon sheaths and slings resulting in joint deformity but, in this case, there is no bone deformity.

Jaccoud's arthropathy is seen in:

- SLE
- Rheumatic fever
- Parkinson's disease, and
- Hypocomplementaemic urticarial vasculitis.

Reactive arthritis is a triad of urethritis, conjunctivitis and sero-negative arthritis with no associated neurological sequelae.

Next Question

A 25-year-old female was referred to a neurologist for investigation into recent episodes of loss of consciousness.

She had had approximately ten episodes in the last month. They appeared to happen when in busy shopping centres or in large crowds and her partner, who had witnessed several, commented that prior to an attack she was somewhat anxious, complained of a headache and her breathing became fast. She then collapsed to the floor motionless for 10 minutes. Her face was red, eyes were closed and on occasions her eyelids flickered. There was no apparent movement of her legs or arms. In one attack, she had been incontinent of urine, but had never bitten her tongue.

Following these episodes she was tearful and upset but appeared orientated. She had never had any nocturnal attacks. She had a history of irritable bowel disease, and had an appendicectomy at 17 years of age with normal histology. She worked as a secretary for a finance company, her partner wants to separate, and she has no contact with her parents.

On examination she appeared fit and well. Her blood pressure was 130/75 mmHg and pulse was 78/min. Heart sounds were normal and chest and abdominal examinations were entirely normal. No abnormalities were found on neurological examination.

ECG	79/min sinus rhythm
Full blood count	Normal
Biochemical profile	Normal
Routine EEG	Normal
CT scan brain	Normal

Given the history and clinical findings, how would you manage this patient?

(Please select 1 option)

- ○ Commence on an anti-depressant
- ○ Arrange a sleep-deprived EEG and MR brain scan
- ○ Arrange EEG and videotelemetry
- ○ Reassure the patient and advise to breathe into a paper bag to abort the attacks
- ○ Commence on lamotrigine 25 mg/day

Submit Answer

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○ Arrange a sleep-deprived EEG and MR brain scan

○ Arrange EEG and videotelemetry **This is the correct answer**

● Reassure the patient and advise to breathe into a paper bag to abort the attacks **Incorrect answer selected**

○ Commence on lamotrigine 25 mg/day

Key Learning Points

Neurology

- Features of NEAD are eye closure, red, or normal colouring, prolonged attacks and post-ictal crying.

Explanation

This patient gives a history of non-epileptic attacks (NEAD).

Clues to NEAD include situational attacks in public and absence of nocturnal events.

There are several features of the attacks which point towards NEAD, including eye closure, red, or normal colouring, prolonged attacks and post-ictal crying. There is a suggestion in the history of previous functional symptoms including appendicectomy with normal histology. Additionally her partner's decision to separate may have led to the onset of her attacks.

Video EEG is the gold standard investigation used to differentiate NEAD from epileptiform events.

Duncan *et al.*[1] write: "Some features traditionally regarded as indicative of epilepsy - such as incontinence of urine and injuries (with the exception of burns) - do not in fact distinguish between epilepsy and Psychogenic Non-Epileptic Seizures."

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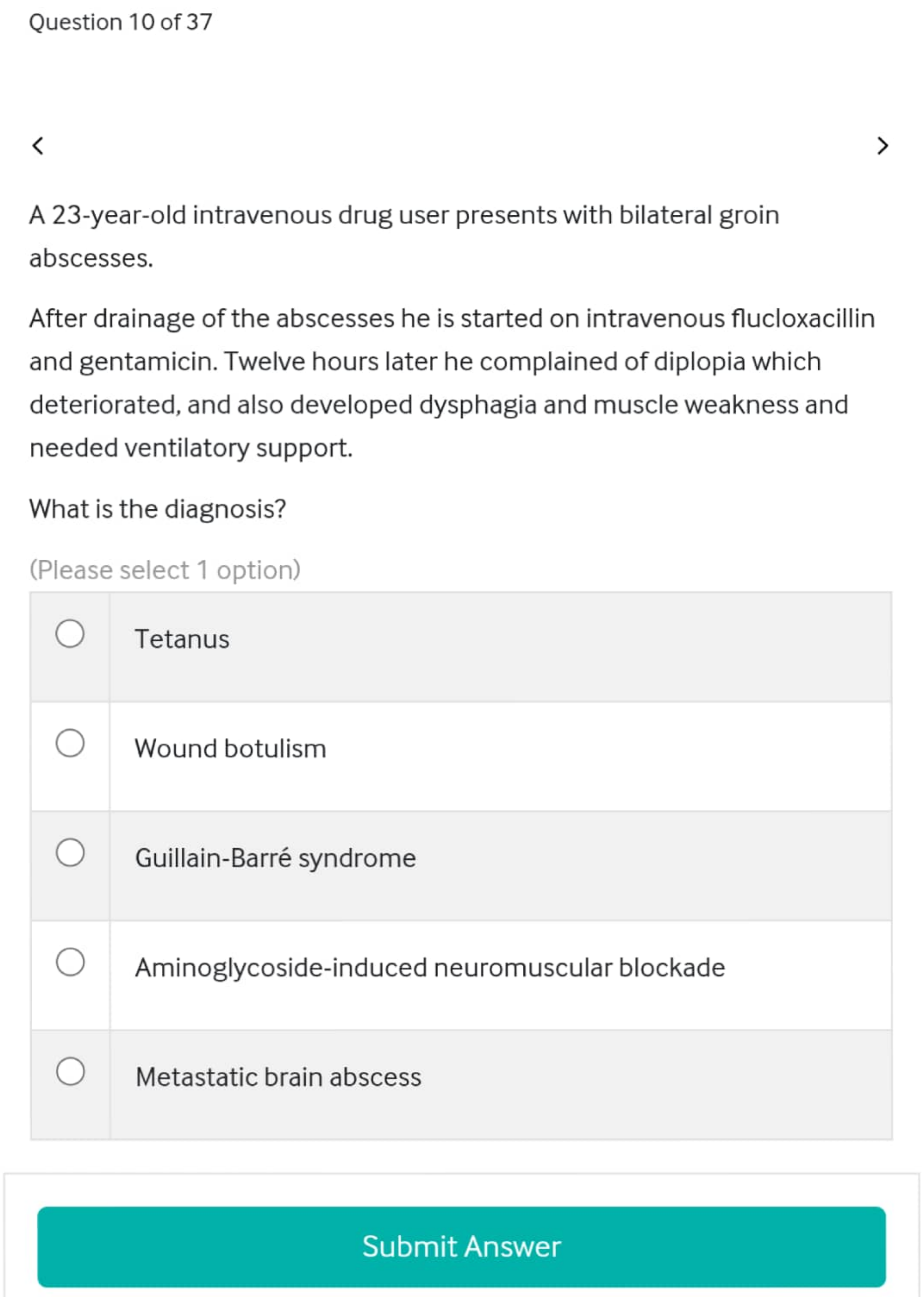

Question 10 of 37

A 23-year-old intravenous drug user presents with bilateral groin abscesses.

After drainage of the abscesses he is started on intravenous flucloxacillin and gentamicin. Twelve hours later he complained of diplopia which deteriorated, and also developed dysphagia and muscle weakness and needed ventilatory support.

What is the diagnosis?

(Please select 1 option)

- ○ Tetanus
- ○ Wound botulism
- ○ Guillain-Barré syndrome
- ○ Aminoglycoside-induced neuromuscular blockade
- ○ Metastatic brain abscess

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○ Guillain-Barré syndrome

● Aminoglycoside-induced neuromuscular blockade Correct

○ Metastatic brain abscess

Key Learning Points

Infectious Diseases, Neurology

- Aminoglycosides may impair neuromuscular transmission and should not be given to patients with myasthenia gravis

Explanation

Aminoglycosides may impair neuromuscular transmission and should not be given to patients with myasthenia gravis; large doses given during surgery have been responsible for a transient myaesthenic syndrome in patients with normal neuromuscular function.

Non-depolarising muscle relaxants (also known as competitive muscle relaxants) compete with acetylcholine for receptor sites at the neuromuscular junction. Non-depolarising muscle relaxants may be divided into the

- Aminosteroid group (for example, pancuronium, vecuronium), and the
- Benzylisoquinolinium group (for example, atracurium).

Wound botulism is the other possible cause but the incubation period for wound botulism is longer, four to 14 days. It would present in a similar way to this case with descending, afebrile, symmetric paralysis particularly affecting the cranial nerves. There is also an absence of gastrointestinal symptoms which are common in botulism.

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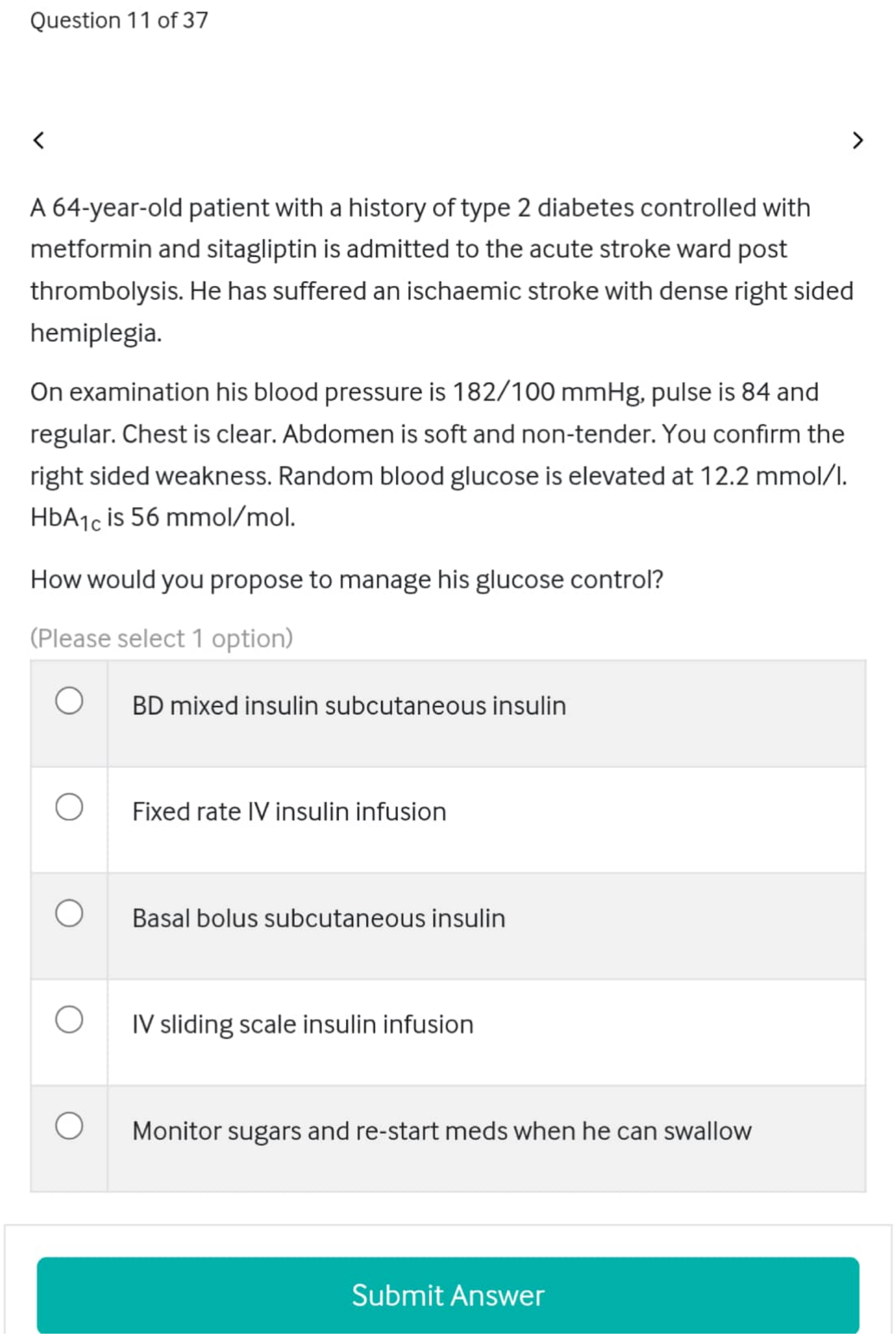


Question 11 of 37

A 64-year-old patient with a history of type 2 diabetes controlled with metformin and sitagliptin is admitted to the acute stroke ward post thrombolysis. He has suffered an ischaemic stroke with dense right sided hemiplegia.

On examination his blood pressure is 182/100 mmHg, pulse is 84 and regular. Chest is clear. Abdomen is soft and non-tender. You confirm the right sided weakness. Random blood glucose is elevated at 12.2 mmol/l. HbA_{1c} is 56 mmol/mol.

How would you propose to manage his glucose control?

(Please select 1 option)

- ○ BD mixed insulin subcutaneous insulin
- ○ Fixed rate IV insulin infusion
- ○ Basal bolus subcutaneous insulin
- ○ IV sliding scale insulin infusion
- ○ Monitor sugars and re-start meds when he can swallow

○ IV sliding scale insulin infusion

● Monitor sugars and re-start meds when he can swallow
Correct

Key Learning Points

Neurology, Stroke

- Diabetic patients with acute stroke resulting in dysphagia should have their oral medication held, sugars monitored and restarted when their swallow improves.

Explanation

The correct answer approach would be to monitor sugars and restart meds when he can swallow.

The subcutaneous insulin options are inappropriate because they may increase risk of hypoglycaemia and over the longer term may drive a worsening in blood pressure and weight gain.

Study evidence suggests intensive insulinisation increases the risk of hypoglycaemia without improving stroke outcomes. Additional evidence from diabetes trials also suggests that hypoglycaemia increases the risk of cardiac arrhythmia.

In this situation, there is no evidence at all to support tight glycaemic control with IV sliding scale insulin or fixed rate IV insulin infusion. A blood sugar of 12.2 mmol/l is elevated but not markedly so, and in keeping with sympathetic drive related to the stroke. His HbaA1c indicates relatively good control.

Next Question

A 45-year-old man presented to the Emergency department with bifrontal headache and nausea which he had had for many weeks.

The pain had steadily worsened over the last 48 hours and he had vomited several times. He had also noticed marked deterioration in his mental capacity. His wife had accompanied him and said he was mentally slowed, easily distractable and his walking had deteriorated.

He had a past medical history of hypertension and took regular bendroflumethiazide. He was a non-smoker and drank 10 units of alcohol per week.

On examination he appeared easily distractable and there was marked perseveration. On cranial nerve examination his pupils were meiotic and fundoscopy revealed bilateral papilloedema. Ocular movements were full and the rest of the cranial nerves were normal. On examination of the upper limb he had bilateral palmomental reflexes, but tone, power and reflexes were all intact. Lower limb examination revealed normal tone, power, reflexes and sensation, but he walked with a somewhat ataxic gait.

A CT scan of the brain showed dilated lateral ventricles with normal-sized third and fourth ventricles.

What is the likely cause of this patient's symptoms?

(Please select 1 option)

○ Periaqueductal gliosis

○ Dandy-Walker syndrome

○ Tumour of the arachnoid villi

○ Tumour at the foramen of Monro

○ Normal pressure hydrocephalus

● Tumour at the foramen of Monro **Correct**

○ Normal pressure hydrocephalus

Key Learning Points

Neurology

- The connection between the lateral ventricles and third ventricle is via the foramen of Monro

Explanation

There is obstruction between the lateral ventricles and third ventricle, as evidenced by the CT scan images.

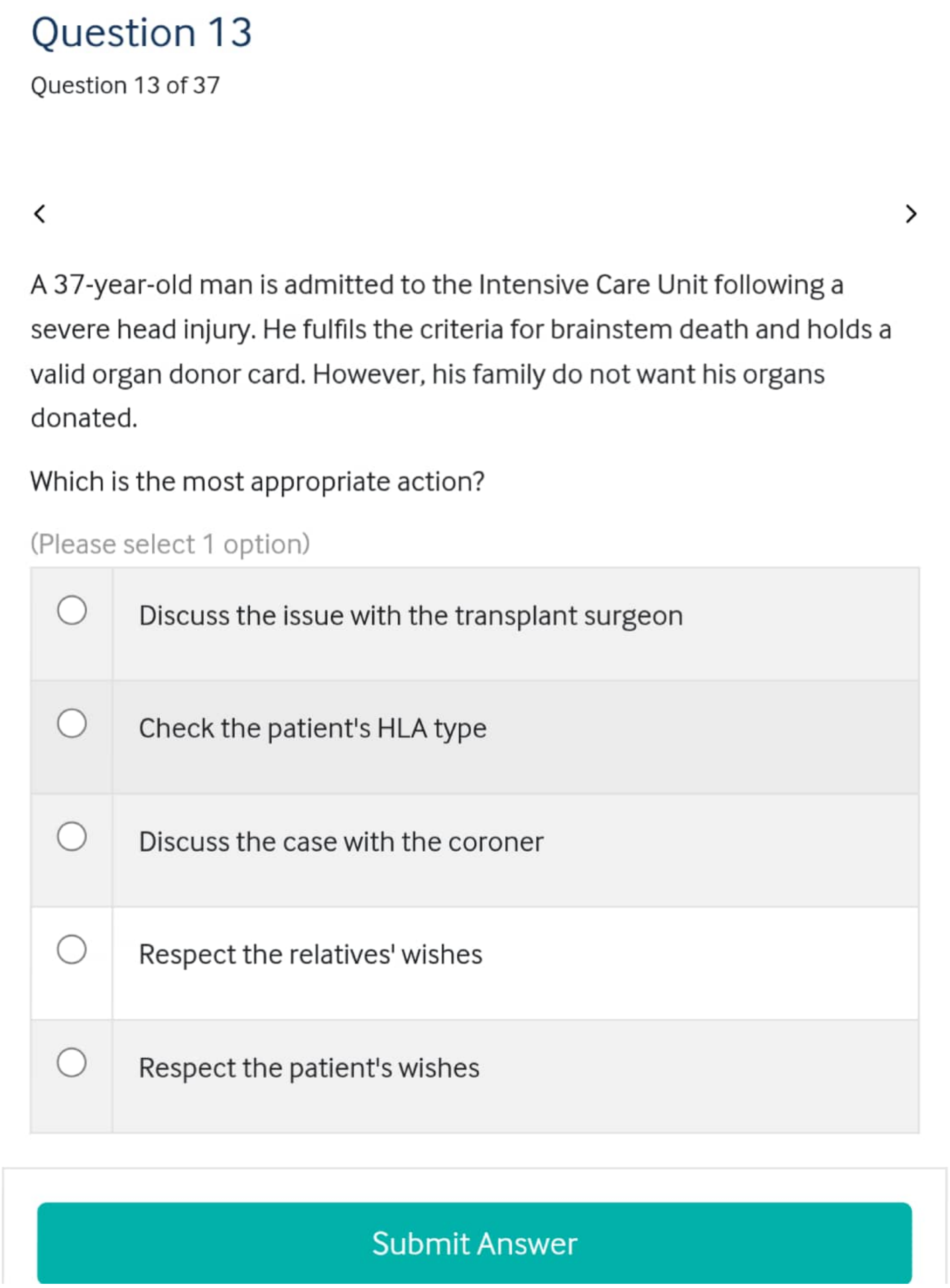
The connection between these sites is via the foramen of Monro, which may be blocked by tumour or can be affected by horizontal shift from an expanding unilateral mass.

Dandy-Walker syndrome is a congenital failure of opening of the foramina of Luschka and Magendie, resulting in dilatation of the entire ventricular system.

The Sylvian aqueduct is the connection between the third and fourth ventricles and if obstructed would cause dilatation of both lateral and third ventricles.

In normal pressure hydrocephalus presenting with gait ataxia, mental dysfunction and sphincteric incontinence, there is dilatation of all the ventricles.

Next Question



Question 13

Question 13 of 37

A 37-year-old man is admitted to the Intensive Care Unit following a severe head injury. He fulfils the criteria for brainstem death and holds a valid organ donor card. However, his family do not want his organs donated.

Which is the most appropriate action?

(Please select 1 option)

○ Discuss the issue with the transplant surgeon

○ Check the patient's HLA type

○ Discuss the case with the coroner

○ Respect the relatives' wishes

○ Respect the patient's wishes

○	Respect the relatives' wishes	This is the correct answer
●	Respect the patient's wishes	Incorrect answer selected

Key Learning Points

Neurology

- The ultimate decision to consent a dead relative to organ "harvest" following the brain-stem death of a patient, rests with the family.

Explanation

In the USA and Europe, only 50% of those who wish to donate organs following their death will have discussed the issue with their family. The family are much more likely to consent to organ donation if these prior discussions have been made and the patient possesses a valid donor card or is on the NHS Organ Donor Register.

If the family objects to the donation, healthcare professionals should discuss the matter tactfully and sensitively with them. They should be encouraged to accept the dead person's wishes. Legally, they do not have the right to reject the patient's wishes, however, the ultimate decision rests with the family and it should be respected.

The most common reasons for relatives' refusal to consent to donation include:

- Did not want surgery to the body
- Patient had previously stated that they did not want to donate
- Patient "had been through enough"
- Family is divided over decision
- Unsure if the patient had wanted to donate, and
- Fear that the body would be disfigured.

The matter should not be discussed with the Transplant Surgeon, as he/she will have a conflict of interest.

There may be reasons why the coroner should be informed of the death of this patient but discussing this particular issue would be inappropriate at this stage.

Human Leukocyte Antigen (HLA) typing will be required once the decision has been made.

Next Question

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A 60-year-old retired builder presented to his GP with a progressive history of stiffness and weakness affecting both legs.

He had recently started dragging his right leg and had noticed some urinary incontinence. His symptoms had come on gradually over a period of four months. There was no history of trauma; however he had had a bout of gastroenteritis in the last few weeks, which he had attributed to eating a take-away curry.

His past medical history included rheumatoid arthritis and tension headaches, which had been more frequent of late, and borderline hypertension. He was a smoker of 20 cigarettes per day and drank 20 units of alcohol per week.

On examination he was alert and orientated. His blood pressure was 142/89 mmHg, pulse 89/min and temperature was 36.7°C. On examination of cranial nerves, no abnormalities were found. On examination of the peripheral nervous system, upper limb was entirely normal, however on examination of the lower limb there was marked spasticity, hyperreflexia with extensor plantar responses. Power was grade 4/5 on the left and 3/5 on the right with a pyramidal pattern of weakness. There did appear to be some sensory neglect of the right lower limb and diminished vibration and light touch on the left lower limb. No sensory level could be detected. There was no cerebellar dysfunction. Chest and abdominal examination was normal.

He was investigated with an MR thoracic spine, which was entirely normal, and lumbar puncture.

Lumbar puncture showed:

Opening pressure	13 cm H_2O	(5-18)
CSF protein	0.6 g/L	(0.15-0.45)
CSF white cell count	20 per ml	(> 5)
CSF red cell count	4 per ml	(>5)
CSF glucose	3.4 mmol/L	(3.3-4.4)
CSF oligoclonal bands	Present	-
Serum oligoclonal bands	Present	-

Given the history and findings so far, what is the likely diagnosis in this patient?

(Please select 1 option)

○ Multiple sclerosis

○ Parasagittal meningioma

○ Guillain-Barré syndrome

○ Central cord syndrome within the cervical spine

○ Anterior spinal artery thrombosis

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● Parasagittal meningioma **Correct**

○ Guillain-Barré syndrome

○ Central cord syndrome within the cervical spine

○ Anterior spinal artery thrombosis

Key Learning Points

Neurology

- Consider parasagittal meningioma with lower limb weakness, cortical sensory loss, bladder dysfunction and raised protein in CSF.

Explanation

This patient presents with a spastic paraparesis in absence of a sensory level, and normal neurology in the upper limb. This places the lesion within the thoracic spine or cerebral cortex. The MR thoracic spine however is normal.

The clues to a probable cortical lesion are sensory neglect in the right leg, headaches and absence of a sensory level.

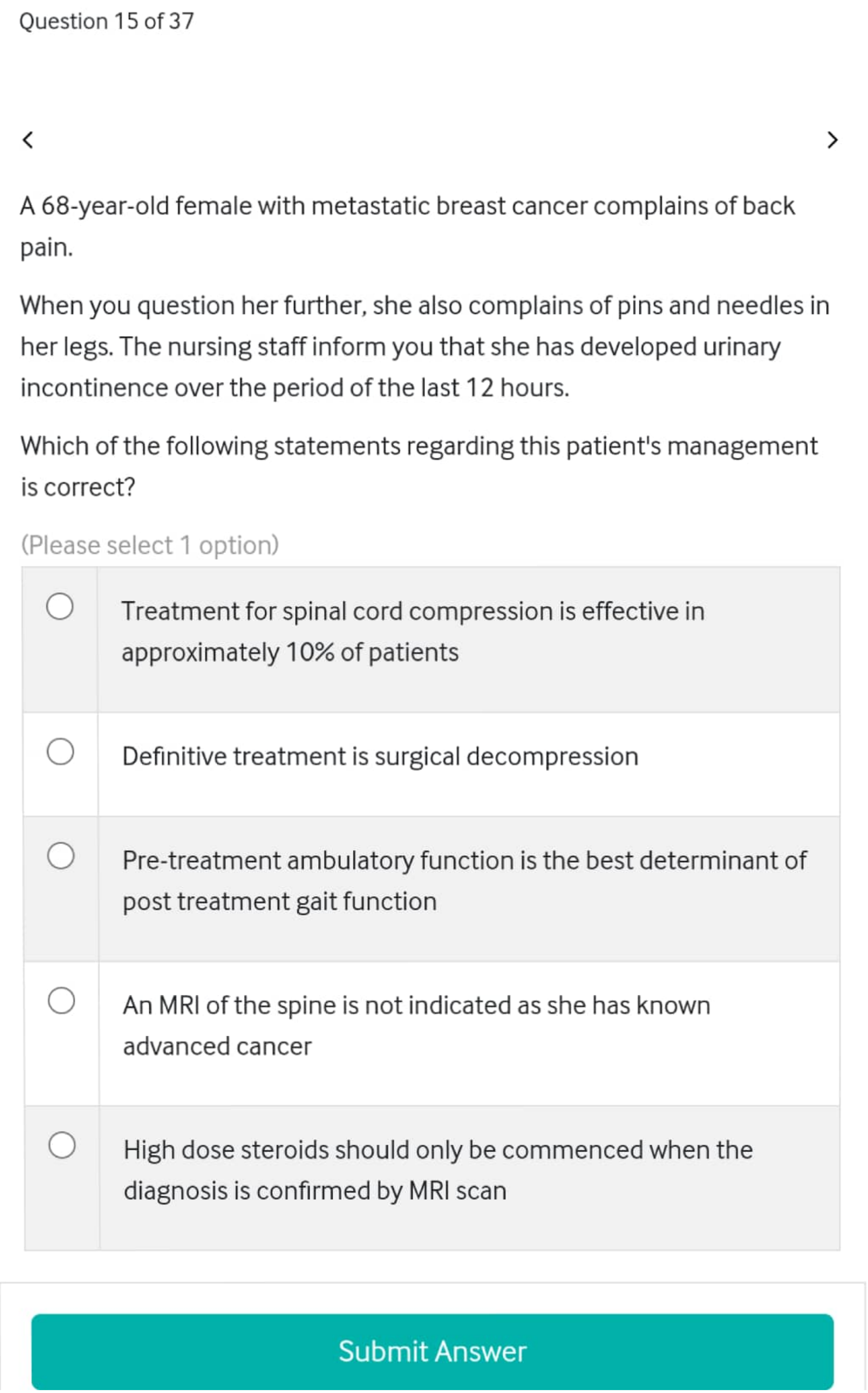
A parasagittal meningioma will primarily cause a weakness of the lower limb and cortical sensory loss. Bladder function may be affected and there may be dysfunction of vibration and light touch sense. The CSF findings of raised protein and WCC are also explained through this.

Multiple sclerosis is unlikely given the age of the patient, normal MR spine and abnormal cerebrospinal fluid (CSF) analysis, which would primarily show intrathecal oligoclonal band production only. The combination of both CSF and serum oligoclonal bands implies a systemic cause for their production, such as rheumatoid arthritis.

Anterior spinal artery thrombosis would cause an acute anterior cord syndrome with dissociated sensory loss affecting the lower limbs.

Guillain-Barré syndrome presents following viral or bacterial enteritis and one would detect lower motor neurone (LMN) signs in the limbs. A central cord syndrome within the cervical spine would cause UMN weakness in the legs, LMN weakness at the level of the lesion, and dissociated sensory loss of pain and temperature in the upper limb.

Next Question



Question 15 of 37

A 68-year-old female with metastatic breast cancer complains of back pain.

When you question her further, she also complains of pins and needles in her legs. The nursing staff inform you that she has developed urinary incontinence over the period of the last 12 hours.

Which of the following statements regarding this patient's management is correct?

(Please select 1 option)

○ Treatment for spinal cord compression is effective in approximately 10% of patients

○ Definitive treatment is surgical decompression

○ Pre-treatment ambulatory function is the best determinant of post treatment gait function

○ An MRI of the spine is not indicated as she has known advanced cancer

○ High dose steroids should only be commenced when the diagnosis is confirmed by MRI scan

○ Pre-treatment ambulatory function is the best determinant of post treatment gait function This is the correct answer

○ An MRI of the spine is not indicated as she has known advanced cancer

● High dose steroids should only be commenced when the diagnosis is confirmed by MRI scan Incorrect answer selected

Key Learning Points

Neurology, Oncology, Palliative Care

- Eighty per cent of patients will maintain mobility if ambulatory function is good at presentation of malignant spinal cord compression.

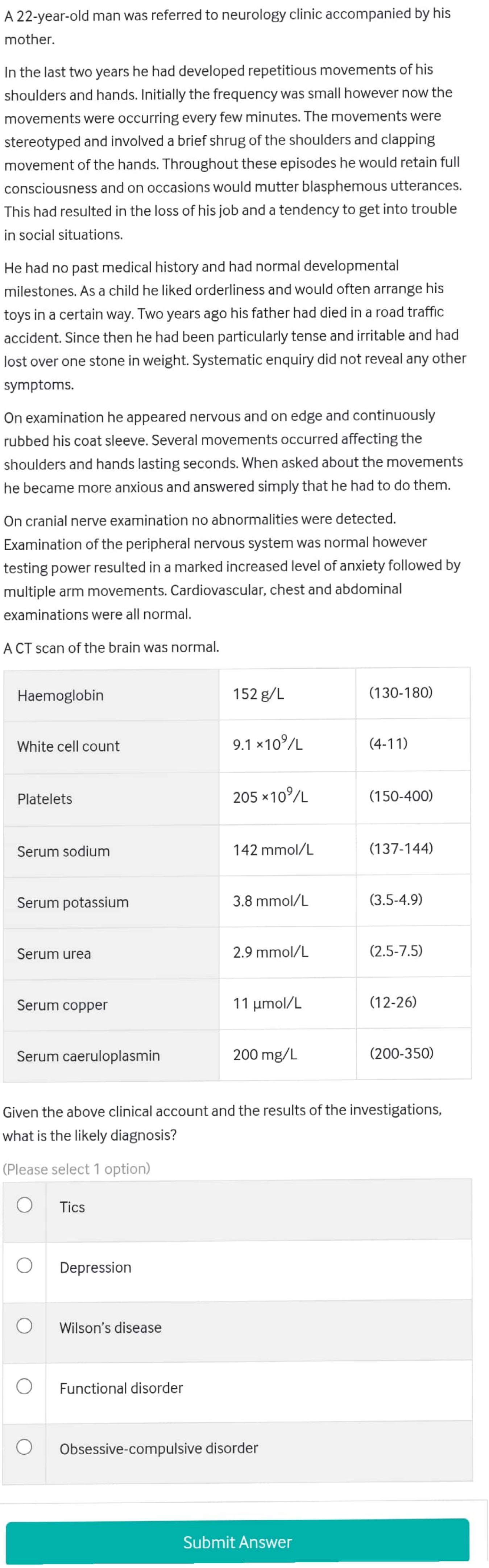
Explanation

This is an oncological emergency. The earlier the diagnosis of spinal cord compression is made, the more likely it is that the treatment will achieve a satisfactory outcome. Eighty per cent of patients will maintain mobility if ambulatory function is good at presentation.

The definitive investigation in this case is an MRI of the vertebral column to look for vertebral collapse or other vertebral disease.

Steroids should be commenced on clinical suspicion of spinal cord compression; diagnosis may be delayed, and steroid treatment may minimise permanent damage.

Urgent radiotherapy is the definitive treatment, although neurosurgical opinion should be sought in order to ensure that surgical decompression is not required. Treatment is effective in 90% of patients if the diagnosis is made early.



A 22-year-old man was referred to neurology clinic accompanied by his mother.

In the last two years he had developed repetitious movements of his shoulders and hands. Initially the frequency was small however now the movements were occurring every few minutes. The movements were stereotyped and involved a brief shrug of the shoulders and clapping movement of the hands. Throughout these episodes he would retain full consciousness and on occasions would mutter blasphemous utterances. This had resulted in the loss of his job and a tendency to get into trouble in social situations.

He had no past medical history and had normal developmental milestones. As a child he liked orderliness and would often arrange his toys in a certain way. Two years ago his father had died in a road traffic accident. Since then he had been particularly tense and irritable and had lost over one stone in weight. Systematic enquiry did not reveal any other symptoms.

On examination he appeared nervous and on edge and continuously rubbed his coat sleeve. Several movements occurred affecting the shoulders and hands lasting seconds. When asked about the movements he became more anxious and answered simply that he had to do them.

On cranial nerve examination no abnormalities were detected. Examination of the peripheral nervous system was normal however testing power resulted in a marked increased level of anxiety followed by multiple arm movements. Cardiovascular, chest and abdominal examinations were all normal.

A CT scan of the brain was normal.

Haemoglobin	152 g/L	(130-180)
White cell count	9.1 $\times 10^9$/L	(4-11)
Platelets	205 $\times 10^9$/L	(150-400)
Serum sodium	142 mmol/L	(137-144)
Serum potassium	3.8 mmol/L	(3.5-4.9)
Serum urea	2.9 mmol/L	(2.5-7.5)
Serum copper	11 µmol/L	(12-26)
Serum caeruloplasmin	200 mg/L	(200-350)

Given the above clinical account and the results of the investigations, what is the likely diagnosis?

(Please select 1 option)

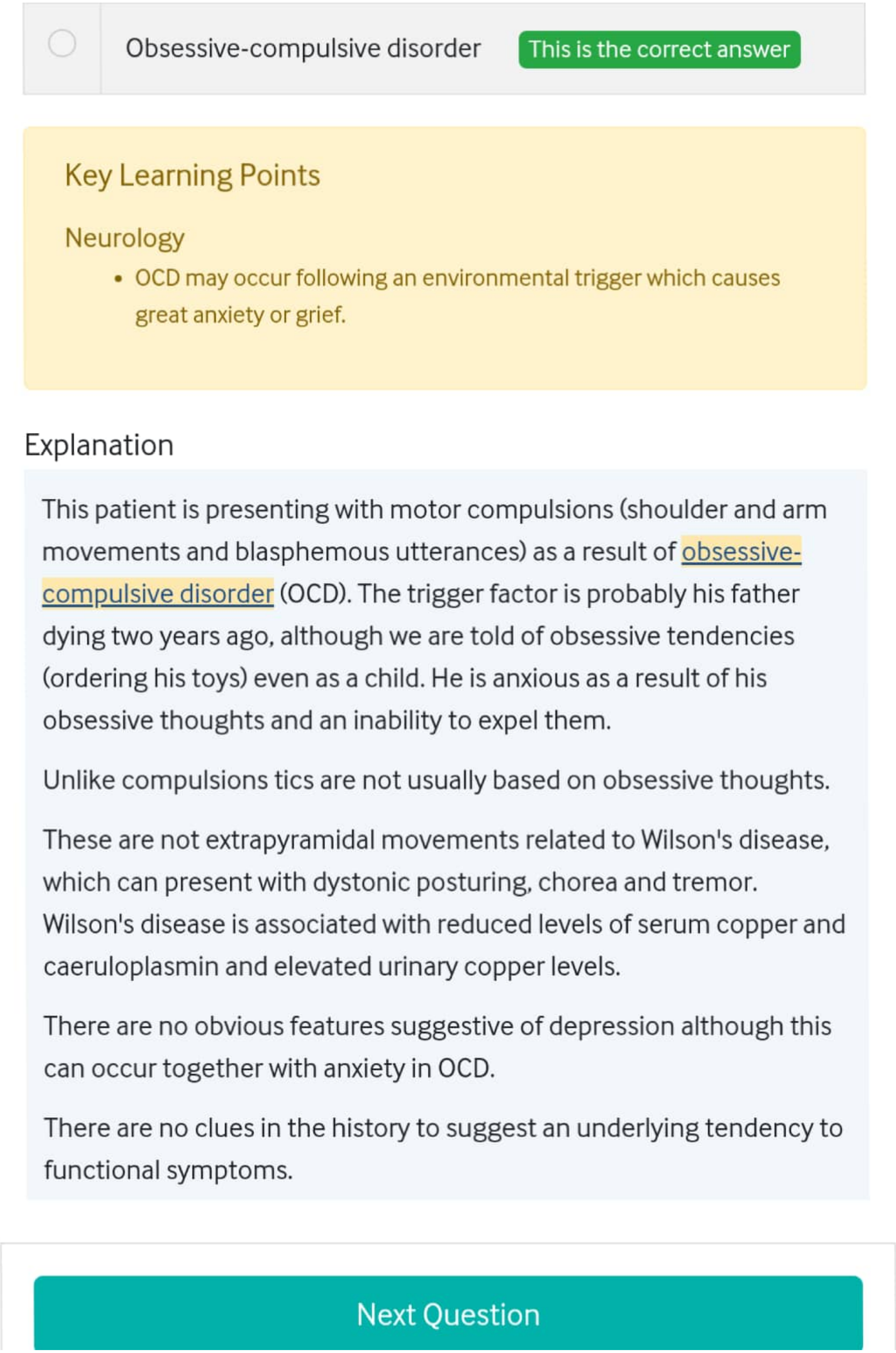
○ Tics

○ Depression

○ Wilson's disease

○ Functional disorder

○ Obsessive-compulsive disorder



○ Obsessive-compulsive disorder **This is the correct answer**

Key Learning Points

Neurology

- OCD may occur following an environmental trigger which causes great anxiety or grief.

Explanation

This patient is presenting with motor compulsions (shoulder and arm movements and blasphemous utterances) as a result of obsessive-compulsive disorder (OCD). The trigger factor is probably his father dying two years ago, although we are told of obsessive tendencies (ordering his toys) even as a child. He is anxious as a result of his obsessive thoughts and an inability to expel them.

Unlike compulsions tics are not usually based on obsessive thoughts.

These are not extrapyramidal movements related to Wilson's disease, which can present with dystonic posturing, chorea and tremor. Wilson's disease is associated with reduced levels of serum copper and caeruloplasmin and elevated urinary copper levels.

There are no obvious features suggestive of depression although this can occur together with anxiety in OCD.

There are no clues in the history to suggest an underlying tendency to functional symptoms.

A 32-year-old woman presented to the Emergency department having developed sudden onset left-sided arm and leg weakness. She had developed a generalised headache prior to the onset of weakness associated with an area of anaesthesia over the right cheek. She had noticed a blurring of vision, but denied any diplopia, visual aura or restriction of visual fields.

She had a past medical history of menorrhagia and had a laparoscopy for intermittent abdominal cramps. She was a smoker and admitted to a cocaine habit. She drank 16 units of alcohol per week, lived with her 4-year-old daughter and did occasional temporary work.

On examination she appeared well and cooperative. Her blood pressure was 135/67 mmHg, pulse was 78/min and regular and heart sounds were normal. There were no carotid bruits. On cranial nerve examination she complained of an area of anaesthesia over the right cheek. Fundoscopy, visual fields, pupillary reflexes and facial musculature were all normal. On examination of the upper limb, there was normal tone bilaterally with brisk symmetrical reflexes. Power was normal in the right arm; however on testing power in the left she was unable to move the arm. Sensation appeared normal. Lower limb examination revealed left sided rigidity with suppressed knee and ankle reflexes and mute plantar response and a power of 0/5.

A contrast-enhanced CT scan of the brain was normal.

A lumbar puncture was performed and yielded the following data:

Opening pressure	13 cmH_2O	(5 - 18)
CSF protein	0.34 g/L	(0.15 - 0.45)
CSF white cell count	4 cells per ml	(<5)
CSF red cell count	3 cells per ml	(<5)
CSF oligoclonal bands	Positive	-
Serum oligoclonal bands	Positive	-

What is the most likely diagnosis in this patient?

(Please select 1 option)

○ Pontine infarct

○ Functional disorder

○ Right carotid artery dissection secondary to cocaine

○ Complex migraine

○ Multiple sclerosis

Submit Answer

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○ Functional disorder This is the correct answer

● Right carotid artery dissection secondary to cocaine Incorrect answer selected

○ Complex migraine

○ Multiple sclerosis

Key Learning Points

Neurology

- Neurological signs and symptoms which vary and lack a pattern of distribution may be suggestive of a functional disorder.

Explanation

This patient presents with a multitude of symptoms and signs which do not fit in with any particular pattern or lesion.

She has complete paralysis of her left arm and leg, but she does not exhibit hyperreflexia or hypertonia. The reflexes are supressed in the left leg probably as a result of sustained rigidity. The area of anaesthesia over the cheek does not fit in with any particular branch of the trigeminal nerve and in absence of other cranial nerve palsies is likely to be insignificant.

The diagnosis is likely to be functional in origin.

There are no associated features or past history of headaches to suggest complex migraine.

Multiple sclerosis can present with odd sensory disturbance although clearly there is no true neurology to be found. The presence of oligoclonal bands in cerebrospinal fluid combined with their absence in blood serum often indicates that immunoglobulins are produced in the central nervous system - in this patient, oligoclonal bands where found in both CSF and serum and is therefore of less significance.

A pontine infarct would cause crossed signs (ipsilateral cranial nerve palsies with contralateral long-tract signs).

A right carotid artery dissection would cause a total anterior circulation stroke with motor and sensory disturbance, homonymous hemianopia and dysphasia, etc.

Next Question

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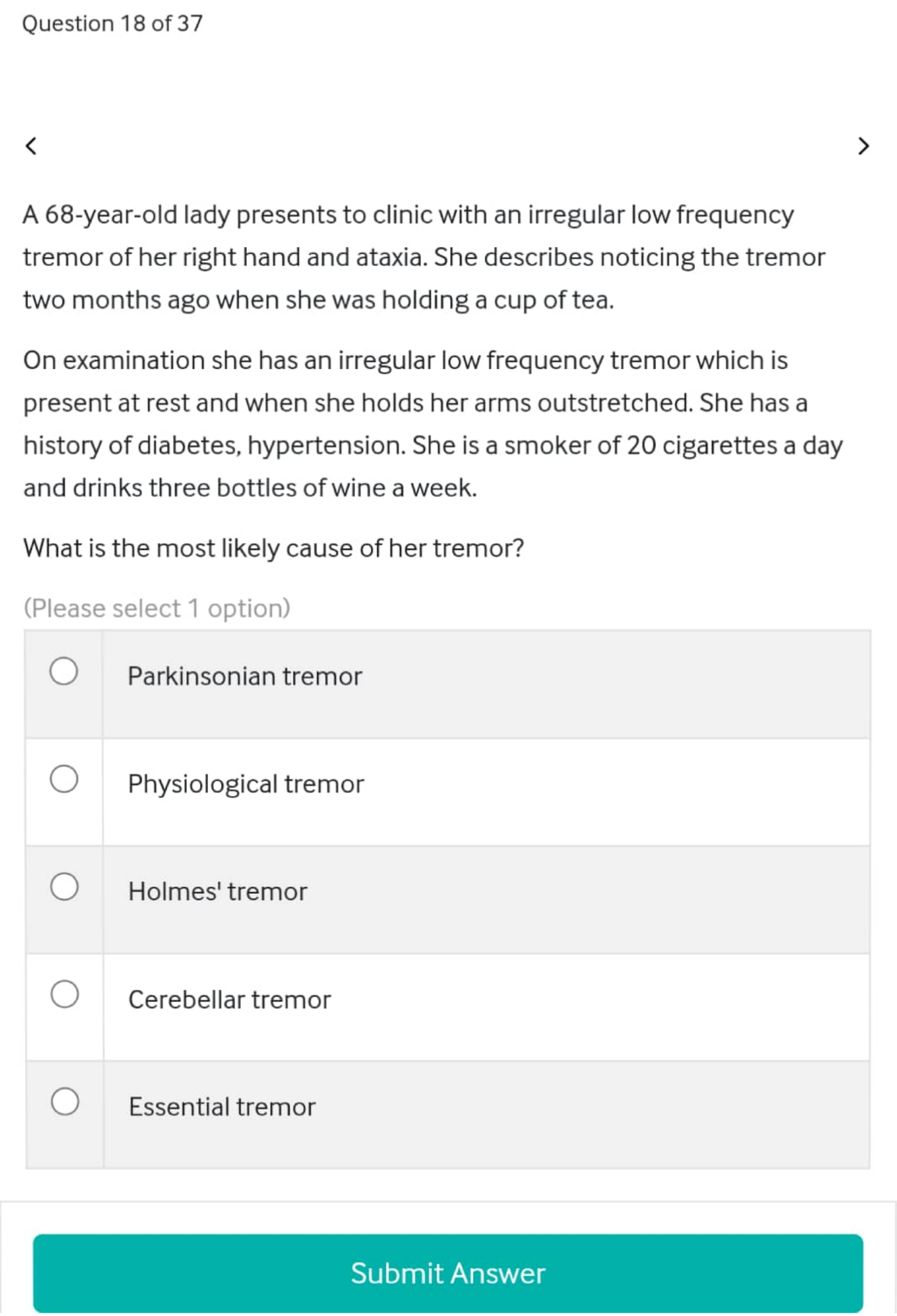

Question 18 of 37

A 68-year-old lady presents to clinic with an irregular low frequency tremor of her right hand and ataxia. She describes noticing the tremor two months ago when she was holding a cup of tea.

On examination she has an irregular low frequency tremor which is present at rest and when she holds her arms outstretched. She has a history of diabetes, hypertension. She is a smoker of 20 cigarettes a day and drinks three bottles of wine a week.

What is the most likely cause of her tremor?

(Please select 1 option)

○ Parkinsonian tremor

○ Physiological tremor

○ Holmes' tremor

○ Cerebellar tremor

○ Essential tremor

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○ Physiological tremor

○ Holmes' tremor This is the correct answer

○ Cerebellar tremor

● Essential tremor Incorrect answer selected

Key Learning Points

Neurology

- Holmes' tremor or rubral tremor is secondary to a lesion in the red nucleus and classically produces an irregular low frequency tremor .

Explanation

Holmes' tremor or rubral tremor is secondary to a lesion in the red nucleus and can occur following a stroke in this area. It classically produces an irregular low frequency tremor which is a combination of a rest, postural and action tremor as described in this case.

It is described as a 'wing-beating' type of tremor and is most prominent when the affected person is active or is maintaining a particular posture. Signs of ataxia and weakness can occur.

This lady has multiple risk factors for cerebrovascular disease including hypertension, diabetes, smoking and excess alcohol use.

Essential tremor typically presents as action tremor which can affect the person's ability to perform activities of daily living.

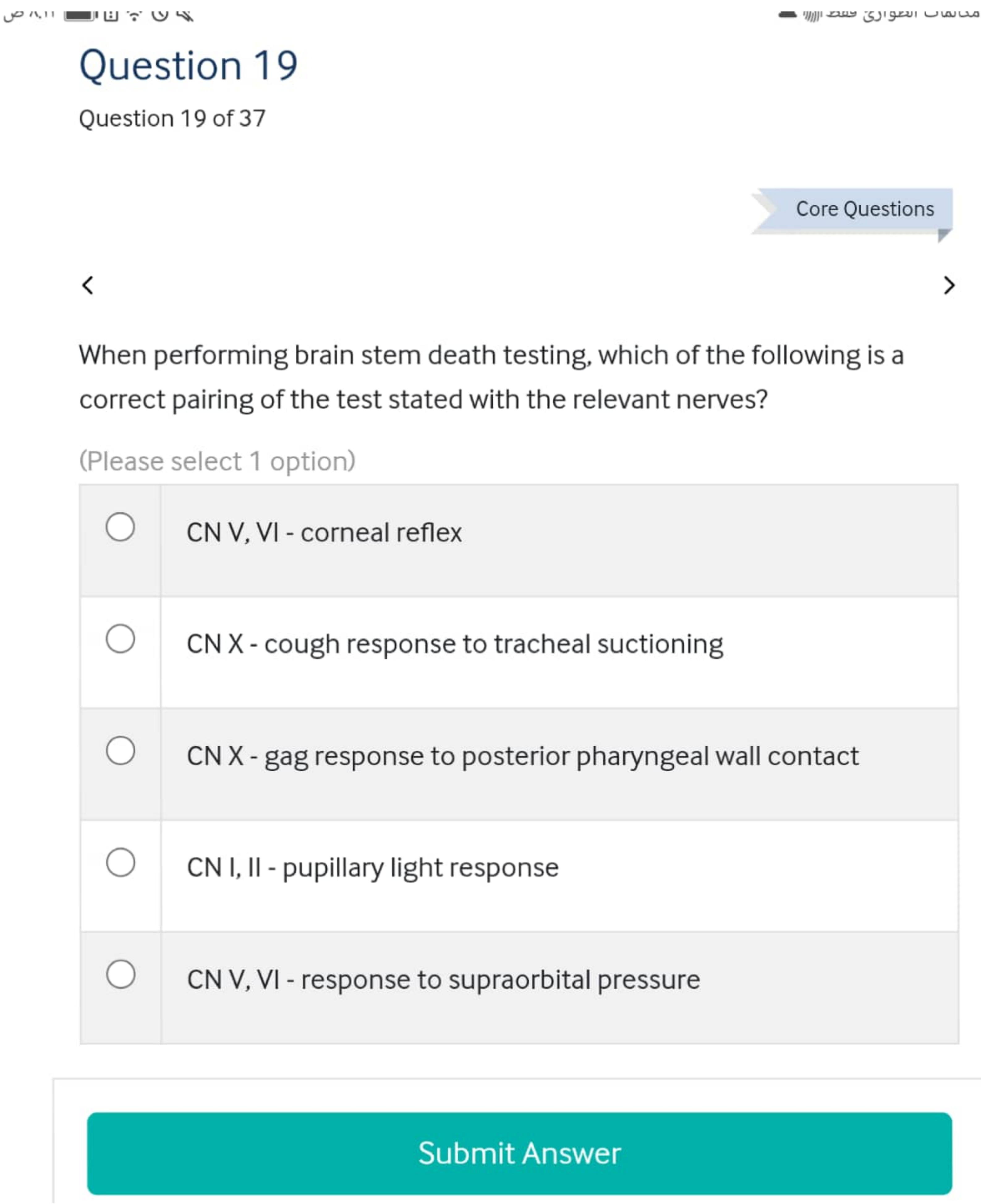
Cerebellar tremor is a slow intention tremor, occurring at the end of a purposeful movement such as touching a finger to the tip of the person's nose. This tremor may be accompanied by dysarthria, nystagmus, gait problems, and postural tremor of the trunk and neck.

Parkinsonian tremor is characteristically an asymmetrical resting tremor. The tremor is classically seen as a 'pill-rolling' action of the hands that may also affect the chin, lips, legs, and trunk, can be markedly increased by stress or emotions.

Physiological tremor is a postural tremor occurring in every normal individual. It is rarely visible to the eye and may be heightened by strong emotion (such as anxiety or fear), physical exhaustion, hypoglycaemia, hyperthyroidism, heavy metal poisoning, stimulants, alcohol withdrawal, caffeine, or fever.

This case describes a tremor which is postural, and occurring with action and rest. Therefore Holmes' tremor is the most likely answer.

Next Question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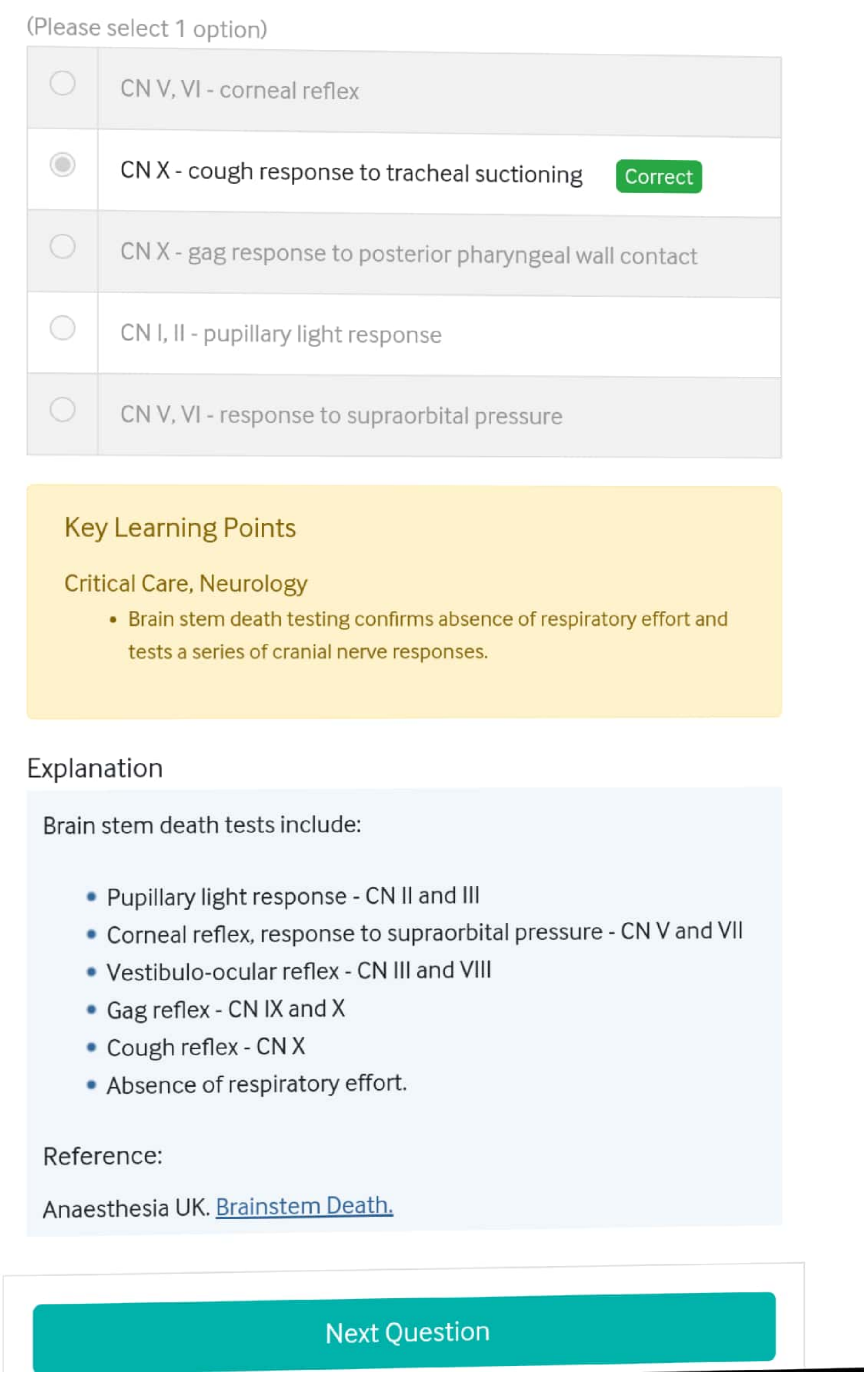
Question 19

Question 19 of 37

When performing brain stem death testing, which of the following is a correct pairing of the test stated with the relevant nerves?

(Please select 1 option)

- ○ CN V, VI - corneal reflex
- ○ CN X - cough response to tracheal suctioning
- ○ CN X - gag response to posterior pharyngeal wall contact
- ○ CN I, II - pupillary light response
- ○ CN V, VI - response to supraorbital pressure



(Please select 1 option)

○ CN V, VI - corneal reflex

● CN X - cough response to tracheal suctioning Correct

○ CN X - gag response to posterior pharyngeal wall contact

○ CN I, II - pupillary light response

○ CN V, VI - response to supraorbital pressure

Key Learning Points

Critical Care, Neurology

- Brain stem death testing confirms absence of respiratory effort and tests a series of cranial nerve responses.

Explanation

Brain stem death tests include:

- Pupillary light response - CN II and III
- Corneal reflex, response to supraorbital pressure - CN V and VII
- Vestibulo-ocular reflex - CN III and VIII
- Gag reflex - CN IX and X
- Cough reflex - CN X
- Absence of respiratory effort.

Reference:

Anaesthesia UK. Brainstem Death.

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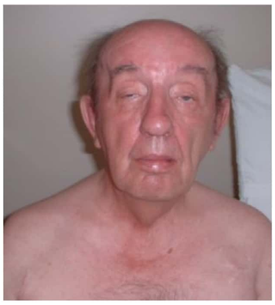
A 58-year-old male was seen in the neurology clinic having been referred with weakness of both hands and wrists. In the preceding week he had been having several falls and his mother, who had accompanied him, felt he was dragging his feet whilst walking.

He had recently been to see his GP because of weight loss and had been diagnosed with impaired glucose tolerance. Otherwise he suffered occasional palpitations, but had no known medical history and did not take any regular medication.



On examination the patient had a monotonous, nasal voice. His general appearance is shown in the picture. Cranial nerve examination revealed bilateral ptosis, but pupils and fundoscopy were intact. There was marked bilateral facial weakness and also some weakness of neck flexion and extension.

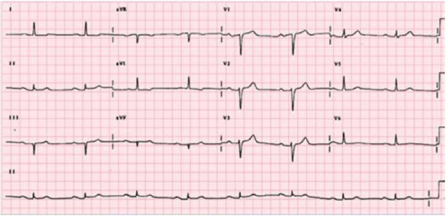
Upper limb inspection revealed marked distal wasting of the hands and forearm musculature. There was reduced tone and supinator jerks bilaterally with weakness of finger flexion and extension and also wrist extension. There was some mild weakness of shoulder abduction bilaterally.

Lower limb examination revealed wasting around the ankles, with reduced ankle tone and reflexes and weakness of dorsiflexion and plantarflexion. Sensory examination was intact.

Investigations revealed:

Haemoglobin	152 g/L	(130-180)
Mean cell volume	93 fL	(80-96)
White cell count	5.1 $\times 10^9$/L	(4-11)
Platelets	221 $\times 10^9$/L	(150-400)
Serum sodium	137 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	6.6 mmol/L	(2.5-7.5)
Serum creatinine	61 mol/L	(60-110)
Serum creatine kinase	170 U/L	(24-170)
Plasma lactate	1.2 mmol/L	(0.6-1.2)
Fasting plasma glucose	8.2 mmol/L	(3-6)
Plasma TSH	4.2 mU/L	(0.5-5.0)
Plasma T4	62 nmol/L	(58-174)
Plasma T3	1.99 nmol/L	(1.07-3.18)

His electrocardiogram is shown below:



How would you investigate this patient?

(Please select 1 option)

- ○ DNA analysis for mitochondrial mutations
- ○ Trial of Sinemet
- ○ Tensilon test
- ○ DNA analysis for mutations in the dystrophin gene
- ○ EMG

Submit Answer

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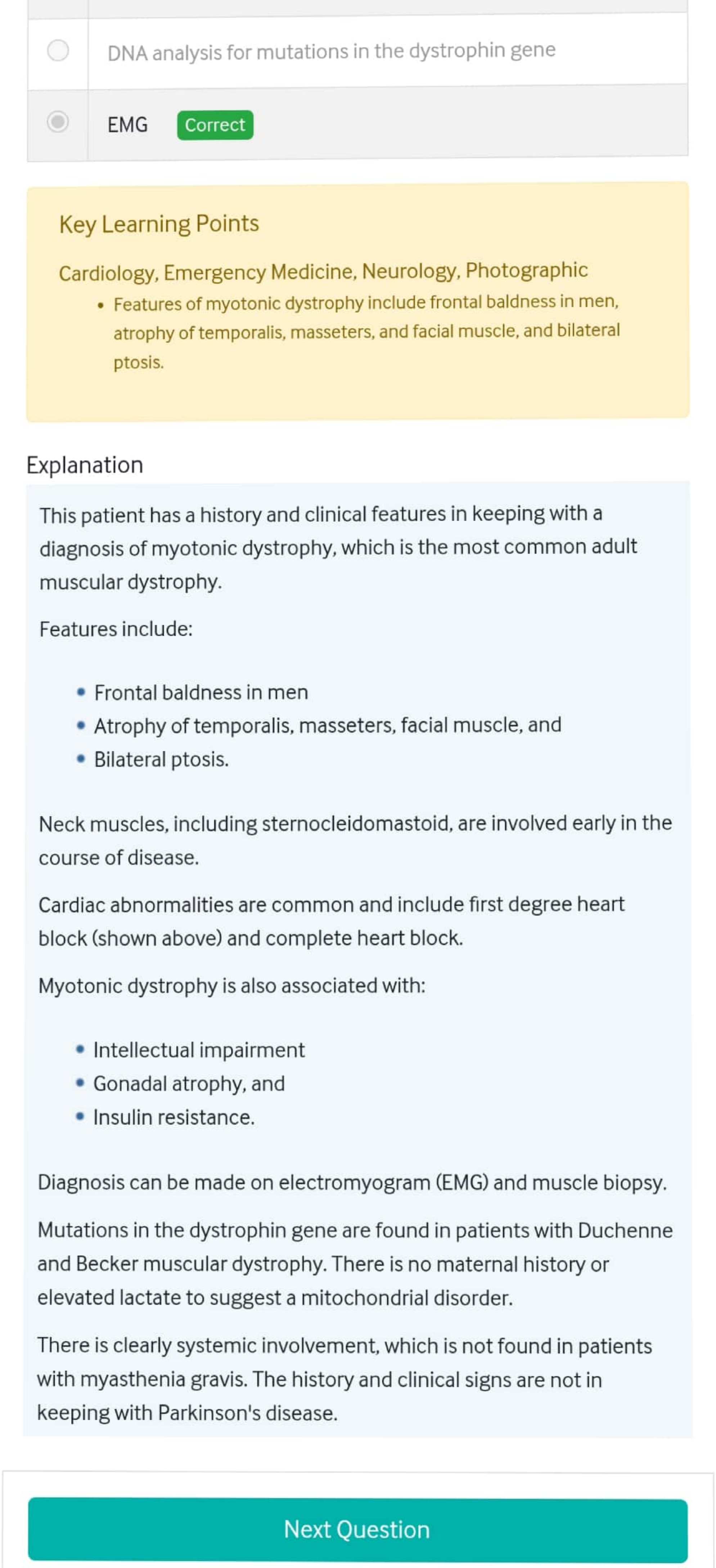

○	DNA analysis for mutations in the dystrophin gene
●	EMG **Correct**

Key Learning Points

Cardiology, Emergency Medicine, Neurology, Photographic

- Features of myotonic dystrophy include frontal baldness in men, atrophy of temporalis, masseters, and facial muscle, and bilateral ptosis.

Explanation

This patient has a history and clinical features in keeping with a diagnosis of myotonic dystrophy, which is the most common adult muscular dystrophy.

Features include:

- Frontal baldness in men
- Atrophy of temporalis, masseters, facial muscle, and
- Bilateral ptosis.

Neck muscles, including sternocleidomastoid, are involved early in the course of disease.

Cardiac abnormalities are common and include first degree heart block (shown above) and complete heart block.

Myotonic dystrophy is also associated with:

- Intellectual impairment
- Gonadal atrophy, and
- Insulin resistance.

Diagnosis can be made on electromyogram (EMG) and muscle biopsy.

Mutations in the dystrophin gene are found in patients with Duchenne and Becker muscular dystrophy. There is no maternal history or elevated lactate to suggest a mitochondrial disorder.

There is clearly systemic involvement, which is not found in patients with myasthenia gravis. The history and clinical signs are not in keeping with Parkinson's disease.

A 45-year-old woman was referred to the neurology outpatient clinic by her general practitioner with a six month history of lethargy and weakness.

When the symptoms first started she had managed to cope reasonably well but had felt the need to go to bed early in the evening. More recently, her symptoms had affected her work and she had lost her job as a result. She had worked in a local supermarket for several years stacking shelves, but had noticed increasing difficulty in lifting boxes. On further questioning, she also reported occasional episodes of double vision over the past two weeks. Her husband, who attended the appointment with her, had been concerned that she might be covertly drinking alcohol to excess as he had noticed that her speech had become slurred when he returned home in the evening.

She had a history of rheumatoid arthritis and was taking regular diclofenac. She was a smoker of 20 cigarettes per day and denied drinking in excess of 10 units of alcohol per week.

On examination she had nasal speech and bilateral ptosis. She was able to breath count to 20. There were reduced eye movements, particularly on left lateral gaze and some facial and neck weakness. There were no obvious neck masses.

Examination of the upper limb revealed normal tone, with marked proximal weakness, which appeared fatigable. There was no associated wasting or fasciculations. Reflexes and sensation appeared preserved. On lower limb examination, there was evidence of weak hip flexion and extension with preserved distal power. Reflexes and sensation were all intact. Chest expansion was 5 cm and cardiovascular examination were both normal.

Investigations revealed:

Anti-acetylcholine receptor antibodies	Negative
Anti-muscle specific tyrosine kinase (MuSK) antibodies	Positive
Tensilon test	Positive

A CT scan of her thorax showed thymic atrophy.

Given the history and clinical findings what would be the most appropriate management for this patient?

(Please select 1 option)

- ○ Commence on pyridostigmine 60 mg QDS
- ○ Commence on intravenous immunoglobulin 0.4 mg/kg/day for five days
- ○ Arrange for plasmaphoresis and high dependency unit transfer
- ○ Commence on pyridostigmine 30 mg QDS and prednisolone 60 mg OD
- ○ Commence on 3, 4-diaminopyridine (3, 4-DAP)

Submit Answer

(Please select 1 option)

● Commence on pyridostigmine 60 mg QDS **Correct**

○ Commence on intravenous immunoglobulin 0.4 mg/kg/day for five days

○ Arrange for plasmaphoresis and high dependency unit transfer

○ Commence on pyridostigmine 30 mg QDS and prednisolone 60 mg OD

○ Commence on 3, 4-diaminopyridine (3, 4-DAP)

Key Learning Points

Neurology

- treatment with IV immunoglobulin or plasmaphoresisin myasthenia gravis are reserved for patients with severe weakness with respiratory involvement.

Explanation

This patient has seronegative myasthenia gravis presenting with ocular, bulbar and limb weakness.

Late onset myasthenia gravis is associated with thymic atrophy as opposed to thymic hyperplasia.

Forty percent of seronegative patients have an antibody found to postsynaptic protein muscle-specific kinase (MuSK).

The most important factor influencing management in this patient is whether there is impending respiratory muscle weakness. This patient had normal chest expansion and breath count, however, formal monitoring of forced vital capacity should be carried out.

In this case, the patient should be initially commenced on pyridostigmine 60 mg QDS. If not responding, prednisolone can be cautiously added in small doses to start with, normally 10-20 mg/day. High dose prednisolone can lead to paradoxical worsening of myasthenia gravis.

IV immunoglobulin or plasmaphoresis are reserved for patients with severe weakness with respiratory involvement.

3, 4-DAP is a treatment used in Eaton-Lambert syndrome where ocular and bulbar weakness is rare and reflexes are generally depressed.

Next Question

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A 58-year-old man was referred to neurology clinic with several months history of increasing bilateral hand weakness and clumsiness (right worse than left).

He had noticed marked difficulty with writing, fine manipulation and had poor hand grip. He had not noticed any sensory disturbance or problems with his neck. He had recently developed some weakness affecting his right knee.

He had a past medical history of hypertension and hypercholesterolaemia and was taking regular bendroflumethiazide and simvastatin. He smoked 20 cigarettes per day and drank 10 units of alcohol per week. There was no family history of medical problems.

On examination, his speech and swallowing appeared intact. Ocular movements were full and the rest of the cranial nerves were normal. General examination of the upper limb revealed bilateral hand and forearm wasting with absent clawing or fasciculations. There also appeared to be some reduced muscle bulk of the proximal musculature. Reflexes were all present and normal.

On examination of power, shoulder abduction/adduction was 3/5 with absent fatigability, and there was marked distal weakness particularly affecting wrist and finger flexors of 2/5, with 3/5 power of finger and wrist extension and 3/5-elbow flexion and extension. Sensation was intact. Lower limb examination revealed some mild weakness of right knee extension (4/5) with normal reflexes, flexor plantar responses and normal sensory examination. Cardiovascular and respiratory examination were both normal.

Investigations revealed:

Haemoglobin	152 g/L	(130 180)
White cell count	7.1 $\times 10^9$/L	(4 11)
Platelets	245 $\times 10^9$/L	(150 400)
Serum sodium	135 mmol/L	(137 144)
Serum potassium	3.6 mmol/L	(3.5 4.9)
Serum urea	5.2 mmol/L	(2.5 7.5)
Serum creatinine	88 µmol/L	(60 110)
Fasting plasma glucose	8.5 mmol/L	(3 6)
Serum cholesterol	4.2 mmol/L	(<5.2)

A lumbar puncture was performed and yielded the following data:

Opening pressure	10 cmH_2O	(5 18)
CSF protein	0.35 g/L	(0.15-0.45)
CSF white cell count	4 cells per ml	(<5)
CSF red cell count	3 cells per ml	(<5)

The chest x ray was normal.

Which of the following investigations would most likely confirm the diagnosis?

(Please select 1 option)

- ○ Nerve conduction studies
- ○ MR cervical spine
- ○ Muscle biopsy
- ○ Anti-ganglioside antibodies
- ○ Electromyography (EMG)

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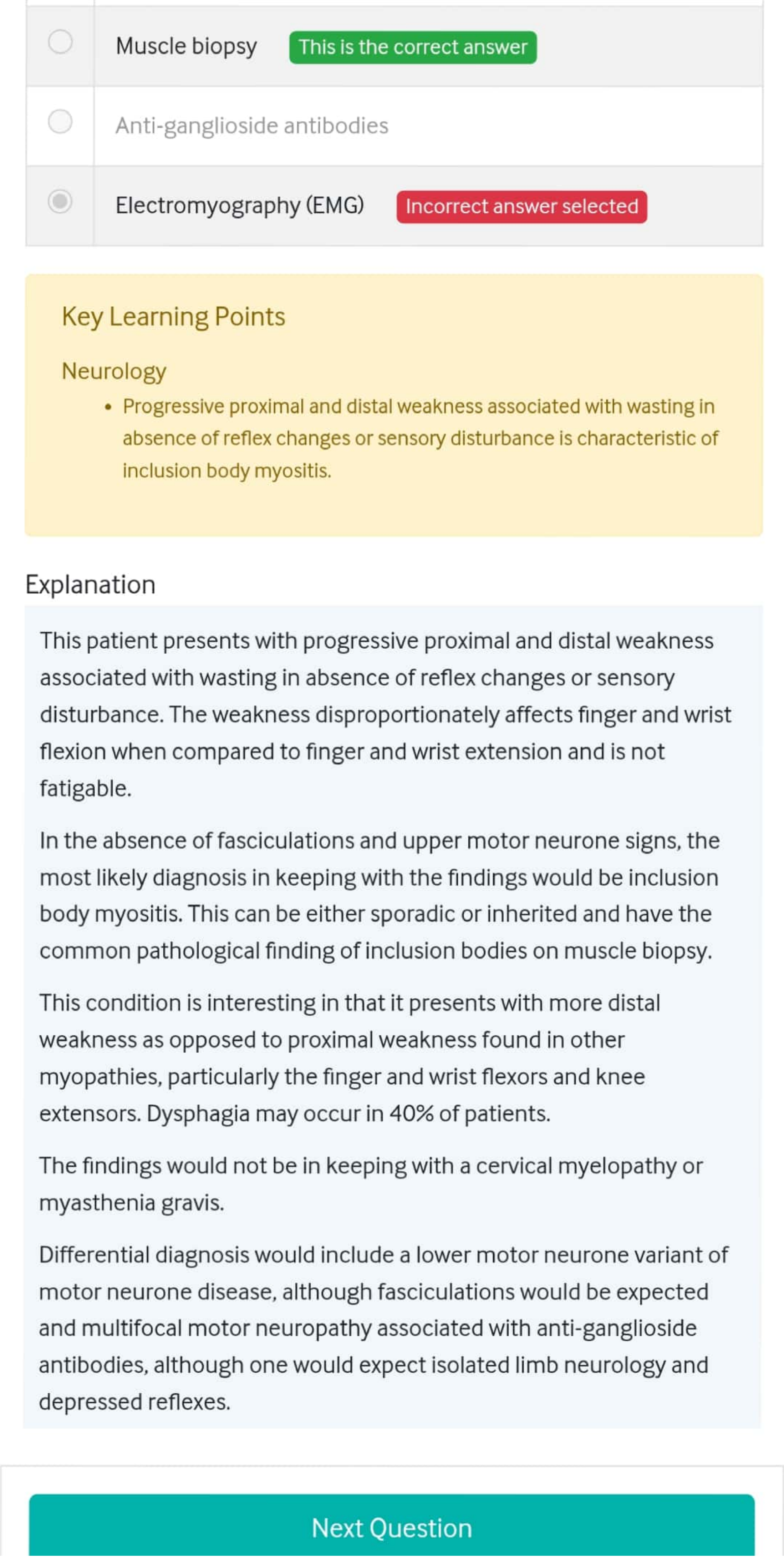

○	Muscle biopsy **This is the correct answer**
○	Anti-ganglioside antibodies
●	Electromyography (EMG) **Incorrect answer selected**

Key Learning Points

Neurology

- Progressive proximal and distal weakness associated with wasting in absence of reflex changes or sensory disturbance is characteristic of inclusion body myositis.

Explanation

This patient presents with progressive proximal and distal weakness associated with wasting in absence of reflex changes or sensory disturbance. The weakness disproportionately affects finger and wrist flexion when compared to finger and wrist extension and is not fatigable.

In the absence of fasciculations and upper motor neurone signs, the most likely diagnosis in keeping with the findings would be inclusion body myositis. This can be either sporadic or inherited and have the common pathological finding of inclusion bodies on muscle biopsy.

This condition is interesting in that it presents with more distal weakness as opposed to proximal weakness found in other myopathies, particularly the finger and wrist flexors and knee extensors. Dysphagia may occur in 40% of patients.

The findings would not be in keeping with a cervical myelopathy or myasthenia gravis.

Differential diagnosis would include a lower motor neurone variant of motor neurone disease, although fasciculations would be expected and multifocal motor neuropathy associated with anti-ganglioside antibodies, although one would expect isolated limb neurology and depressed reflexes.

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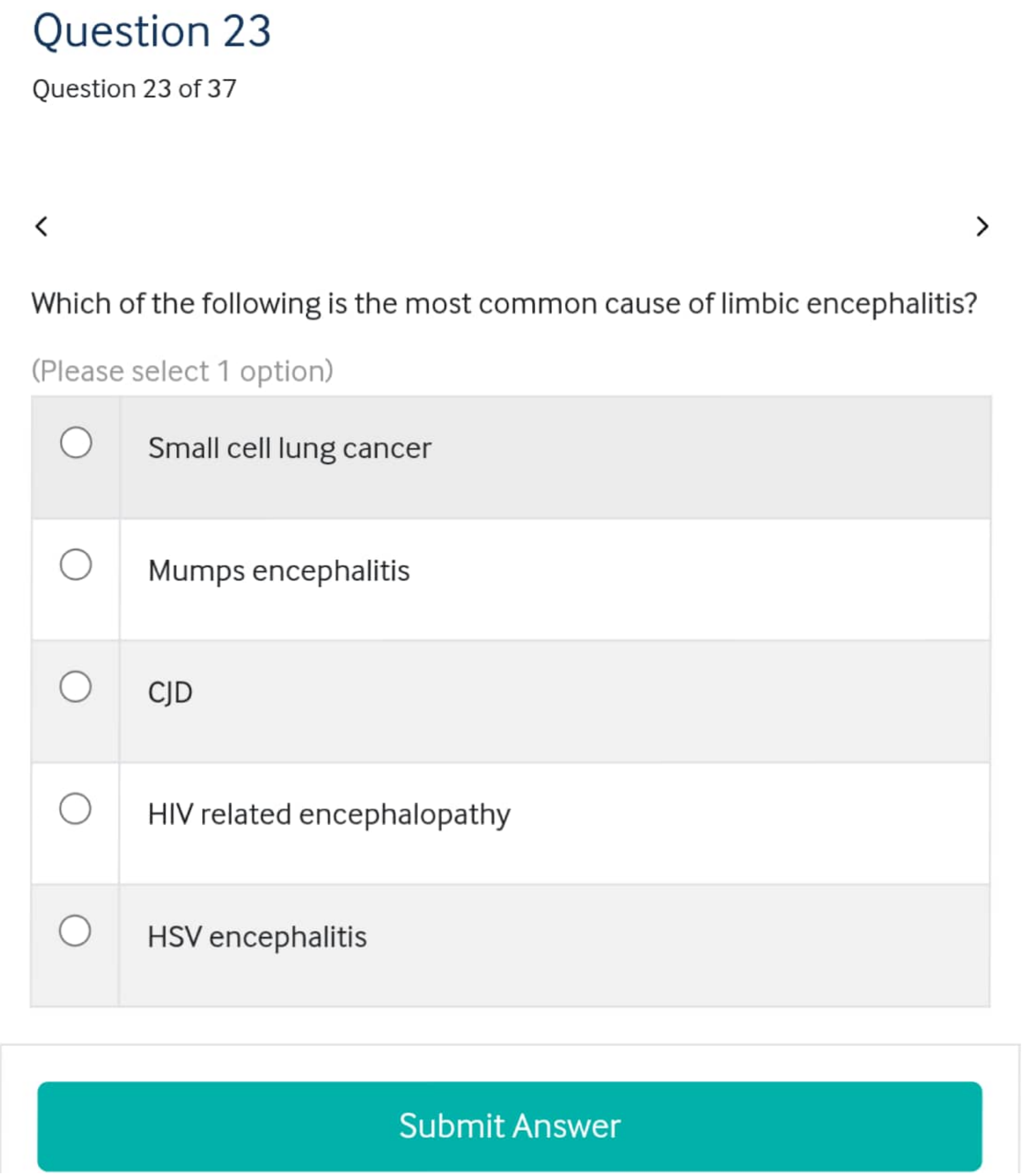

Question 23

Question 23 of 37

Which of the following is the most common cause of limbic encephalitis?

(Please select 1 option)

○ Small cell lung cancer

○ Mumps encephalitis

○ CJD

○ HIV related encephalopathy

○ HSV encephalitis

(Please select 1 option)

○ Small cell lung cancer **This is the correct answer**

● Mumps encephalitis **Incorrect answer selected**

○ CJD

○ HIV related encephalopathy

○ HSV encephalitis

Key Learning Points

Neurology

- Small cell lung cancer commonly presents with paraneoplastic syndromes such as limbic encephalitis.

Explanation

Paraneoplastic neurological syndromes are uncommon but important because they frequently present before the malignancy, and because they cause severe neurological disability. Examples of these are:

- Limbic encephalitis
- Cerebellar degeneration
- Opsoclonus-myoclonus
- Sensory neuronopathy
- Lambert-Eaton myasthenic syndrome
- Myasthenia gravis
- Dermatomyositis, and
- Polymyositis.[1]

Acute to subacute onset of short term memory deficits, with relative preservation of other cognitive functions, is characteristic of limbic encephalitis.[2] The memory deficits may be noticed after several weeks of depression, personality change, or irritability.

Seizures can occur and are most often partial complex seizures. Olfactory and gustatory hallucinations are common.

Some patients also develop signs of diencephalic-hypothalamic dysfunction, including drowsiness, hyperthermia, hyperphagia, and, less frequently, pituitary hormonal deficits.[3]

In 60% of cases, limbic encephalitis is a paraneoplastic disorder and indicates the presence of an underlying cancer; the most common underlying malignancy is small cell lung carcinoma (SCLC), followed by testicular cancer, thymoma, and Hodgkin's lymphoma.[4]

In contrast to patients with other paraneoplastic neurologic syndromes, in whom magnetic resonance imaging (MRI) is of limited usefulness in helping to establish the diagnosis, patients with limbic encephalitis may present with early MRI changes suggestive of the disorder.[5]

Typically, the MRI shows hyperintense abnormalities in the medial aspect of the temporal lobes.[6]

These MRI abnormalities should be differentiated from those in patients with herpes simplex encephalitis, in whom the MRI usually shows signs of oedema, mass effect, contrast enhancement, and, sometimes, areas of haemorrhage.

Regardless of the tumour type, the neurologic dysfunction usually precedes the diagnosis of cancer.

Most paraneoplastic syndromes respond poorly to immunomodulatory treatment although occasional improvement is seen when the underlying tumour is treated.

Differential diagnosis of acute/subacute encephalopathy is etiologically wide and includes:

- Neurodegenerative (for example sporadic Creutzfeldt-Jakob disease [CJD])
- Endocrine (hypothyroidism)
- Toxicological (lead, arsenic poisoning)
- Nutritional (vitamin B1 deficiency)
- Infective (HSV, HIV), and
- Autoimmune causes.[7]

Due to the diversity of symptoms of limbic encephalitis, this disorder is likely underdiagnosed. In patients without a known cancer, limbic encephalitis may be mistaken for a viral encephalitis or a rapidly developing dementia secondary to a neurodegenerative disorder.[8]

Typical symptoms of limbic encephalitis described above supported by MRI findings, in association with cerebrospinal fluid (CSF) changes characteristic of inflammation are highly suggestive of paraneoplastic limbic encephalitis and should prompt testing for antineuronal antibodies.

Among patients with SCLC, the anti-Hu antibody is present in about 50% of those with predominant or isolated symptoms of limbic encephalitis.[4,9]

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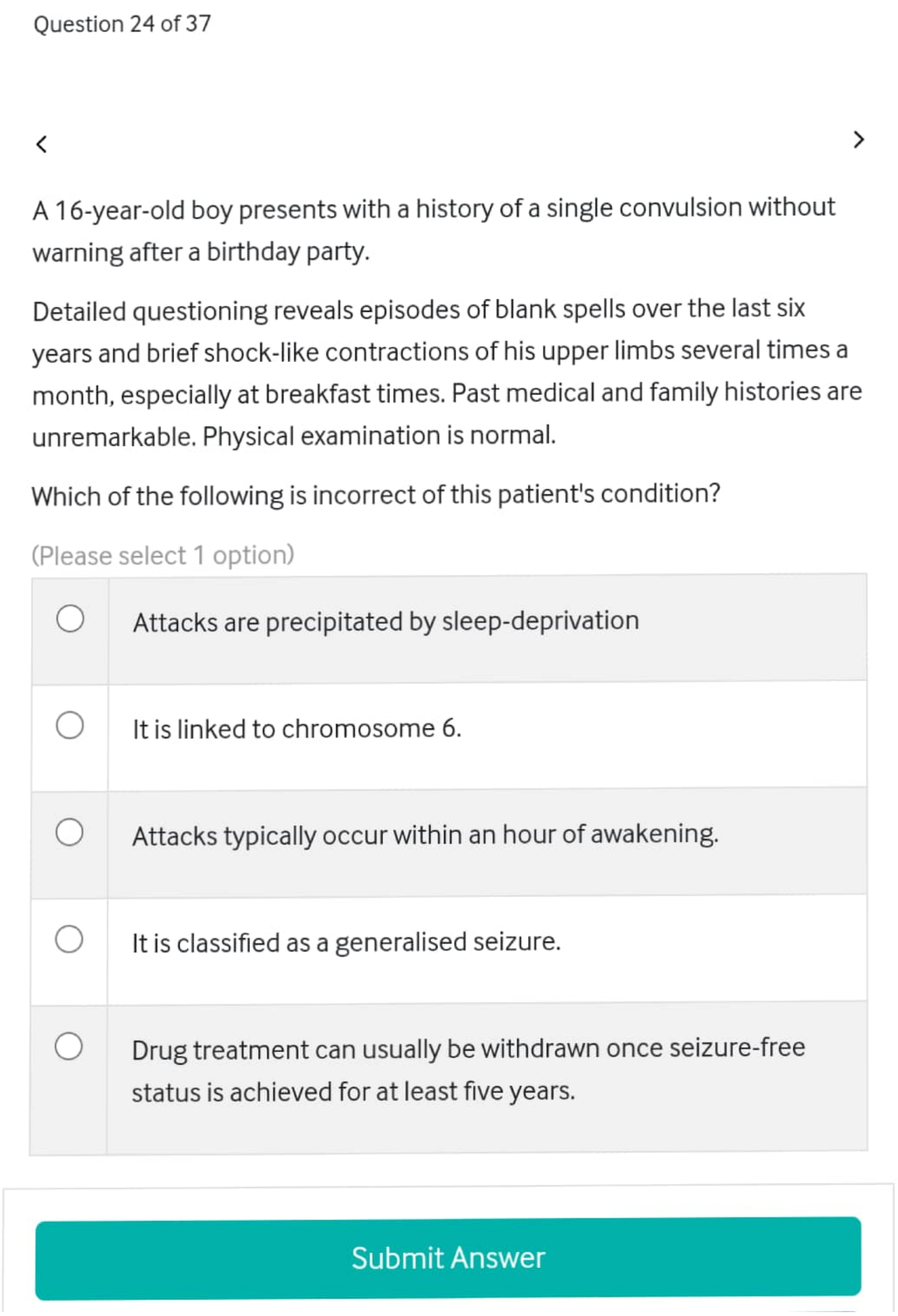


Question 24 of 37

A 16-year-old boy presents with a history of a single convulsion without warning after a birthday party.

Detailed questioning reveals episodes of blank spells over the last six years and brief shock-like contractions of his upper limbs several times a month, especially at breakfast times. Past medical and family histories are unremarkable. Physical examination is normal.

Which of the following is incorrect of this patient's condition?

(Please select 1 option)

○ Attacks are precipitated by sleep-deprivation

○ It is linked to chromosome 6.

○ Attacks typically occur within an hour of awakening.

○ It is classified as a generalised seizure.

○ Drug treatment can usually be withdrawn once seizure-free status is achieved for at least five years.

CamScanner الممسوحة ضوئيا بـ

○ It is classified as a generalised seizure.

● Drug treatment can usually be withdrawn once seizure-free status is achieved for at least five years. Correct

Key Learning Points

Neurology

- Juvenile myoclonic epilepsy is associated with absences, myoclonic jerks and tonic-clonic seizures which tend to occur within an hour of waking.

Explanation

Juvenile myoclonic epilepsy is the commonest of the idiopathic generalised epilepsies.

Seizures types include

- Absences
- Myoclonic jerks and
- Tonic-clonic seizures which tend to occur within an hour of waking.

Precipitating factors include alcohol, menstruation and sleep deprivation.

The condition is genetically linked to the short arm of chromosome 6.

Prognosis is extremely favourable if the condition is treated correctly, with many patients becoming seizure-free.

Treatment options include sodium valproate, lamotrigine and topiramate.

Lifelong drug treatment is usually necessary to avoid relapses in patients who achieve seizure-free status on medication.

CamScanner الممسوحة ضوئيا بـ

A 35-year-old woman was admitted to the Emergency department having developed restlessness, hallucinations and acute confusion. The symptoms came on within minutes following the consumption of sumatriptan for one of her migraines.

On admission she was vomiting and sweating profusely and appeared confused, with a Glasgow coma scale of 14/15. Her blood pressure was 162/90 mmHg, pulse was 102 beats per minute and regular and temperature was 38.9°C. Heart sounds were normal and chest was clear on auscultation.

Cranial nerve examination was normal including fundoscopy. Peripheral nervous system examination revealed normal tone with four beats of ankle clonus bilaterally. Reflexes were globally brisk however power, sensation and coordination were all normal.

She was taking fluoxetine for depression and sumatriptan for occasional migraines. She was a non-smoker and drank 10 units of alcohol per week.

Investigations revealed:

Sodium	135 mmol/L	(137-144)
Potassium	3.9 mmol/L	(3.5-4.9)
Urea	8.9 mmol/L	(2.5-7.5)
Creatinine	130 µmol/L	(60-110)
Haemoglobin	102 g/L	(115-165)
White cell count	12×10^9/L	(4-11)
Platelets	100×10^9/L	(150-400)
Prothrombin time	20.2 s	(11.5-15.5)
Activated partial thromboplastin time	50 s	(30-40)
Fibrinogen	0.8 g/L	(1.8-5.4)

Which one of these potential therapeutic options should the patient be given?

(Please select 1 option)

○	Diazepam
○	Dantrolene
○	Cyproheptadine
○	IV fluids
○	bicarbonate

○ Dantrolene

● Cyproheptadine Correct

○ IV fluids

○ bicarbonate

Key Learning Points

Neurology

- Cyproheptadine treats serotonin syndrome

Explanation

This patient has developed serotonin syndrome together with disseminated intravascular coagulation (low platelets and fibrinogen, high prothrombin time, and activated partial thromboplastin time) as a result of sumatriptan consumption in addition to her fluoxetine, both of which increase serotonin levels.

Serotonin toxicity leads to changes in mental status, systemic changes and motor complications.

Changes in mental status include:

- confusion
- agitation
- hypomania
- anxiety, and
- coma.

Systemic changes include:

- tachycardia
- hypertension
- nausea
- salivation, and
- Pyrexia.

Motor complications include:

- myoclonus
- hyper-reflexia
- seizures
- in-coordination, and
- tremor.

Muscle rigidity can occur but is more likely in neuroleptic malignant syndrome.

Treatment is supportive and involves removing the precipitating drugs, controlling agitation, administering serotonin antagonists (cyproheptadine or methysergide) and controlling hyperthermia.

Dantrolene is a drug used in neuroleptic malignant syndrome for controlling muscle rigidity which is not present in this patient.

Next Question

<   >

A 60-year-old woman presented with a three month history of diplopia and blurred vision of left eye. She denied any pain or other neurological symptoms. Her previous medical history is unremarkable. She smokes 20 cigarettes per day and drinks alcohol in moderation.

Her general medical examination is normal. Her visual acuity on the right is 6/6 and on the left 6/36. There is left partial ptosis and mild proptosis with conjunctival injection. The left pupil is smaller than the right but reacting normally to light. There is some limitation of abduction of the left eye. Fundoscopy showed a pale left optic disc. The left corneal reflex is reduced. The remainder of the neurological examination is normal.

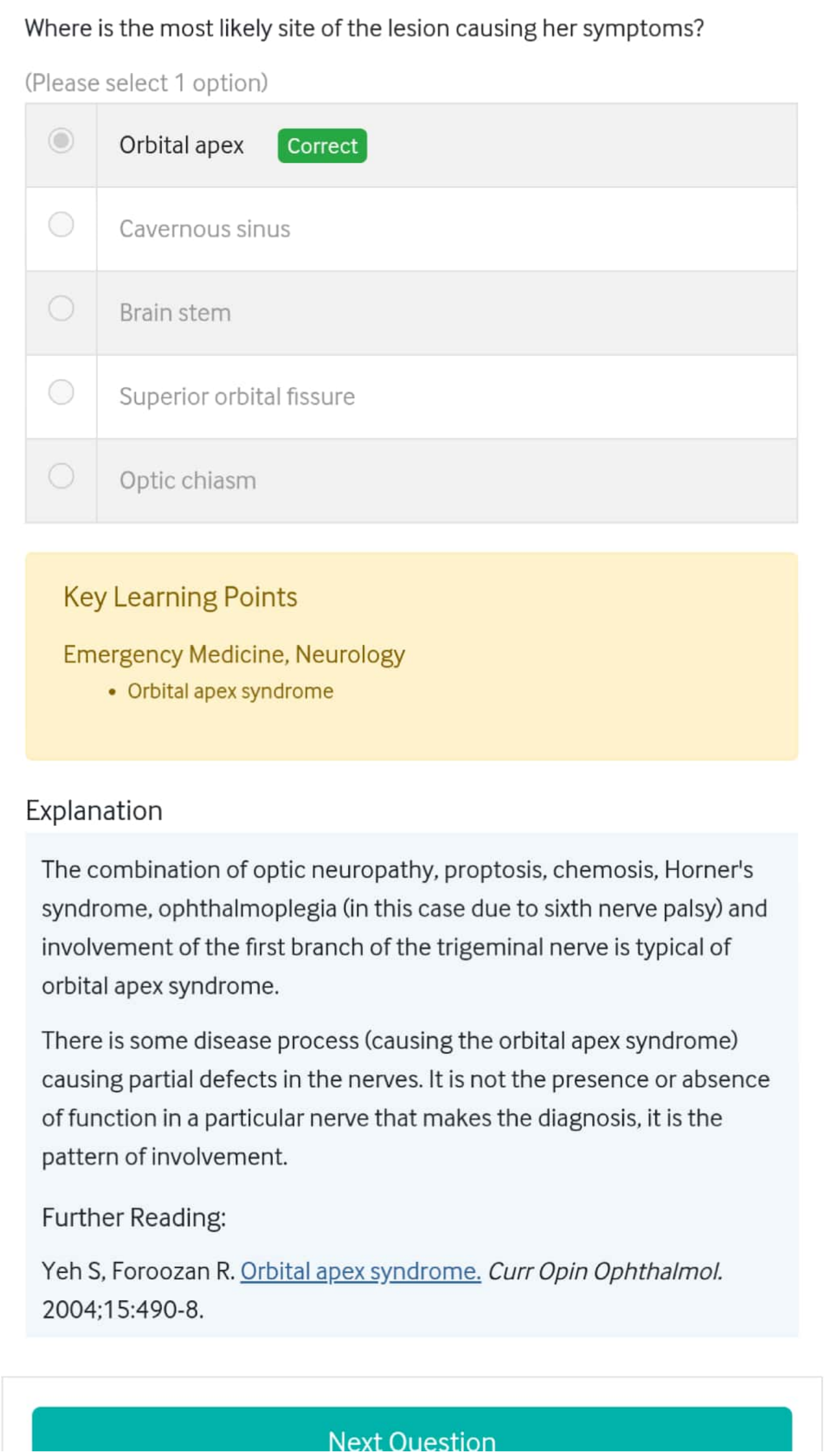
Routine blood tests including full blood count, urea and electrolytes, liver function tests, thyroid function, serum calcium, serum creatine kinase, and autoantibody screen were normal. Her electrocardiogram and chest radiograph showed no abnormalities.

Slit lamp examination was normal and intraocular pressures were within normal range.

Where is the most likely site of the lesion causing her symptoms?

(Please select 1 option)

○	Orbital apex
○	Cavernous sinus
○	Brain stem
○	Superior orbital fissure
○	Optic chiasm



Where is the most likely site of the lesion causing her symptoms?

(Please select 1 option)

- ● Orbital apex **Correct**
- ○ Cavernous sinus
- ○ Brain stem
- ○ Superior orbital fissure
- ○ Optic chiasm

Key Learning Points

Emergency Medicine, Neurology

- Orbital apex syndrome

Explanation

The combination of optic neuropathy, proptosis, chemosis, Horner's syndrome, ophthalmoplegia (in this case due to sixth nerve palsy) and involvement of the first branch of the trigeminal nerve is typical of orbital apex syndrome.

There is some disease process (causing the orbital apex syndrome) causing partial defects in the nerves. It is not the presence or absence of function in a particular nerve that makes the diagnosis, it is the pattern of involvement.

Further Reading:

Yeh S, Foroozan R. Orbital apex syndrome. *Curr Opin Ophthalmol.* 2004;15:490-8.

A 48-year-old woman was seen in neurology clinic with a progressive history of numbness and unsteadiness affecting both feet. She had also noticed wasting of her lower legs and her skin had become shiny and tight. She had been steadily losing weight for many months and had had to give up her job as a secretary due to increasing lethargy.

She had a past medical history of irritable bowel disease, hypothyroidism and had been under the care of a dermatologist in the past for a vesicular skin rash.

On examination she was pale and thin. Examination of the cranial nerves revealed no abnormalities. Upper limb examination revealed normal tone, power and reflexes with preserved sensation. Lower limb examination revealed wasting and atrophic skin changes over both anterior compartments of the lower leg. Tone was bilaterally reduced at the ankle and knee and both plantar responses were mute. Reflexes were diminished at the ankle and knee bilaterally. There was some weakness of dorsiflexion and plantar flexion and she walked with a markedly ataxic gait. Romberg's test was positive. Sensory examination revealed reduced sensation to all modalities extending to both knees. Abdominal examination revealed a tympanic abdomen with no palpable masses.

Investigations showed:

Haemoglobin	96 g/L	(115 - 165)
Mean cell volume	105 fL	(80 - 96)
White cell count	4.2 ×10^9/L	(4 - 11)
Platelets	235 ×10^9/L	(150 - 400)
Serum sodium	133 mmol/L	(137 - 144)
Serum potassium	3.5 mmol/L	(3.5 - 4.9)
Serum urea	5.2 mmol/L	(2.5 - 7.5)
Serum creatinine	106 µmol/L	(60 - 110)
Cholesterol	2.7 mmol/L	(< 5.2)
Serum calcium	1.79 mmol/L	(2.2 - 2.6)
Albumin	23 g/L	(37 - 49)
Serum alkaline phosphatase	127 IU/L	(45 - 105)
Prothrombin time	15.9 seconds	(11.5 - 15.5)
Thyroid stimulating hormone	1.20 mU/L	(0.4 - 5.0)
Thyroxine	169 nmol/L	(58 - 174)
Glucose	4.9 mmol/L	(3.0 - 6.0)
ESR (Westergren)	5 mm/1st hr	-

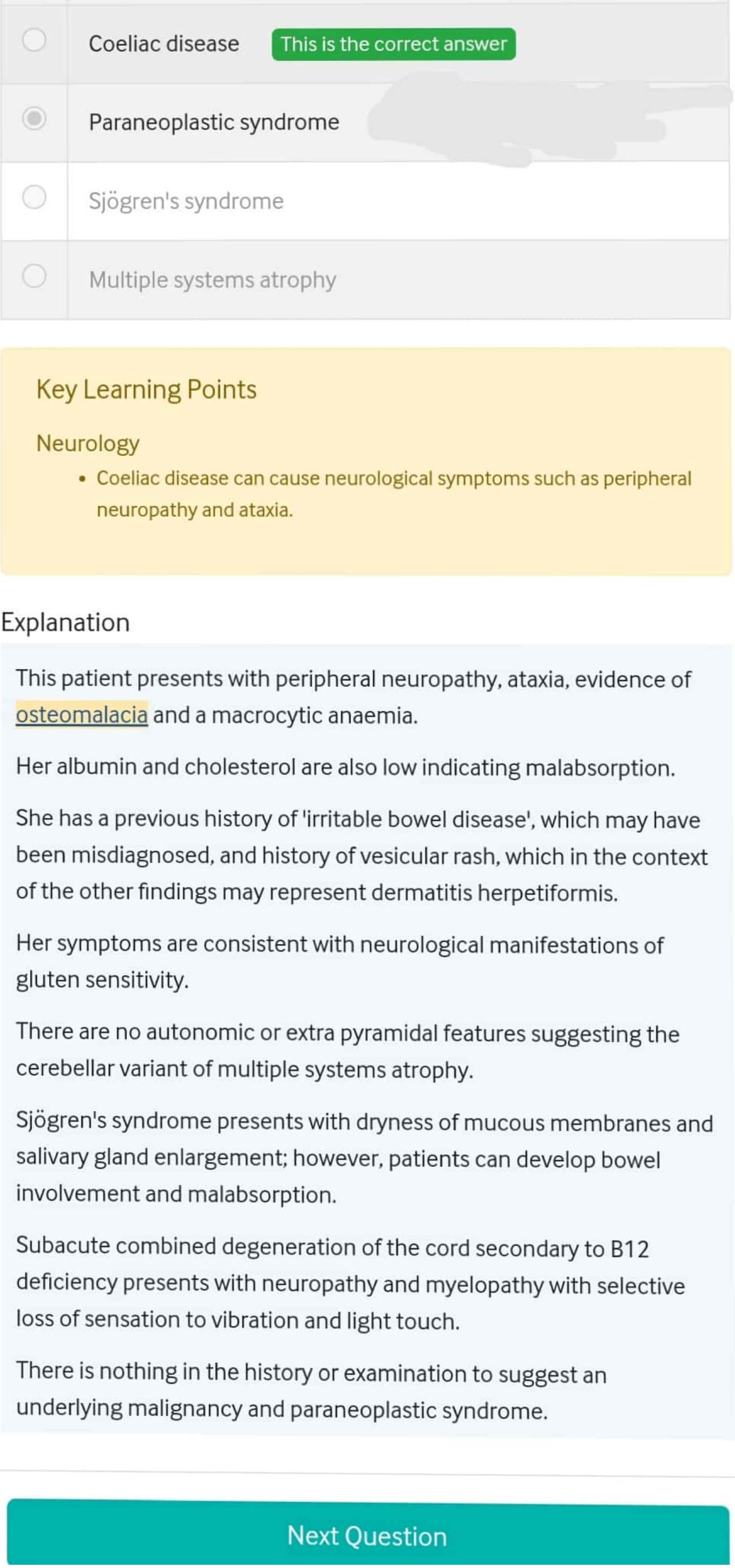
An MRI scan of the brain showed cerebellar atrophy and scattered white matter changes. Nerve conduction studies showed a sensorimotor axonal neuropathy.

What is the underlying diagnosis in this patient?

(Please select 1 option)

- ○ Subacute combined degeneration of the cord
- ○ Coeliac disease
- ○ Paraneoplastic syndrome
- ○ Sjögren's syndrome
- ○ Multiple systems atrophy

Submit Answer



○ Coeliac disease **This is the correct answer**

● Paraneoplastic syndrome [redacted]

○ Sjögren's syndrome

○ Multiple systems atrophy

Key Learning Points

Neurology

- Coeliac disease can cause neurological symptoms such as peripheral neuropathy and ataxia.

Explanation

This patient presents with peripheral neuropathy, ataxia, evidence of osteomalacia and a macrocytic anaemia.

Her albumin and cholesterol are also low indicating malabsorption.

She has a previous history of 'irritable bowel disease', which may have been misdiagnosed, and history of vesicular rash, which in the context of the other findings may represent dermatitis herpetiformis.

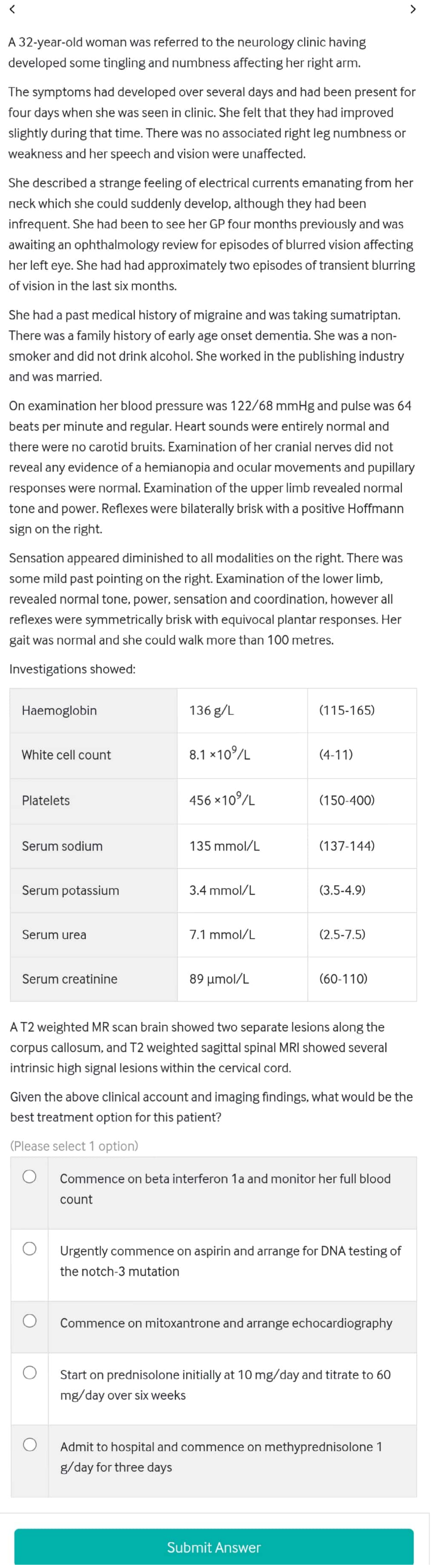
Her symptoms are consistent with neurological manifestations of gluten sensitivity.

There are no autonomic or extra pyramidal features suggesting the cerebellar variant of multiple systems atrophy.

Sjögren's syndrome presents with dryness of mucous membranes and salivary gland enlargement; however, patients can develop bowel involvement and malabsorption.

Subacute combined degeneration of the cord secondary to B12 deficiency presents with neuropathy and myelopathy with selective loss of sensation to vibration and light touch.

There is nothing in the history or examination to suggest an underlying malignancy and paraneoplastic syndrome.



A 32-year-old woman was referred to the neurology clinic having developed some tingling and numbness affecting her right arm.

The symptoms had developed over several days and had been present for four days when she was seen in clinic. She felt that they had improved slightly during that time. There was no associated right leg numbness or weakness and her speech and vision were unaffected.

She described a strange feeling of electrical currents emanating from her neck which she could suddenly develop, although they had been infrequent. She had been to see her GP four months previously and was awaiting an ophthalmology review for episodes of blurred vision affecting her left eye. She had had approximately two episodes of transient blurring of vision in the last six months.

She had a past medical history of migraine and was taking sumatriptan. There was a family history of early age onset dementia. She was a non-smoker and did not drink alcohol. She worked in the publishing industry and was married.

On examination her blood pressure was 122/68 mmHg and pulse was 64 beats per minute and regular. Heart sounds were entirely normal and there were no carotid bruits. Examination of her cranial nerves did not reveal any evidence of a hemianopia and ocular movements and pupillary responses were normal. Examination of the upper limb revealed normal tone and power. Reflexes were bilaterally brisk with a positive Hoffmann sign on the right.

Sensation appeared diminished to all modalities on the right. There was some mild past pointing on the right. Examination of the lower limb, revealed normal tone, power, sensation and coordination, however all reflexes were symmetrically brisk with equivocal plantar responses. Her gait was normal and she could walk more than 100 metres.

Investigations showed:

Haemoglobin	136 g/L	(115-165)
White cell count	8.1 ×10^9/L	(4-11)
Platelets	456 ×10^9/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.4 mmol/L	(3.5-4.9)
Serum urea	7.1 mmol/L	(2.5-7.5)
Serum creatinine	89 µmol/L	(60-110)

A T2 weighted MR scan brain showed two separate lesions along the corpus callosum, and T2 weighted sagittal spinal MRI showed several intrinsic high signal lesions within the cervical cord.

Given the above clinical account and imaging findings, what would be the best treatment option for this patient?

(Please select 1 option)

○ Commence on beta interferon 1a and monitor her full blood count

○ Urgently commence on aspirin and arrange for DNA testing of the notch-3 mutation

○ Commence on mitoxantrone and arrange echocardiography

○ Start on prednisolone initially at 10 mg/day and titrate to 60 mg/day over six weeks

○ Admit to hospital and commence on methyprednisolone 1 g/day for three days

(Please select 1 option)

● Commence on beta interferon 1a and monitor her full blood count Correct

○ Urgently commence on aspirin and arrange for DNA testing of the notch-3 mutation

○ Commence on mitoxantrone and arrange echocardiography

○ Start on prednisolone initially at 10 mg/day and titrate to 60 mg/day over six weeks

○ Admit to hospital and commence on methyprednisolone 1 g/day for three days

Key Learning Points

Neurology

- Beta-interferon can reduce the relapse rate by approximately one third for the first two years of treatment in relapse remitting MS.

Explanation

This patient has relapsing-remitting multiple sclerosis (MS) both clinically and paraclinically with separate clinical episodes disseminated through space and time.

She fulfills the following criteria from the Association of British Neurologists (ABN) for commencing beta-interferon therapy:

- Has had more than two separate episodes within the last two years
- Is more than 18-years-old, and
- Can walk more than 100 metres.

Contraindications to beta-interferon are:

- History of severe clinical depression
- Uncontrolled epilepsy
- Hepatic dysfunction, and
- Myelosupression.

Beta-interferon can reduce the relapse rate by approximately one third for the first two years of treatment.

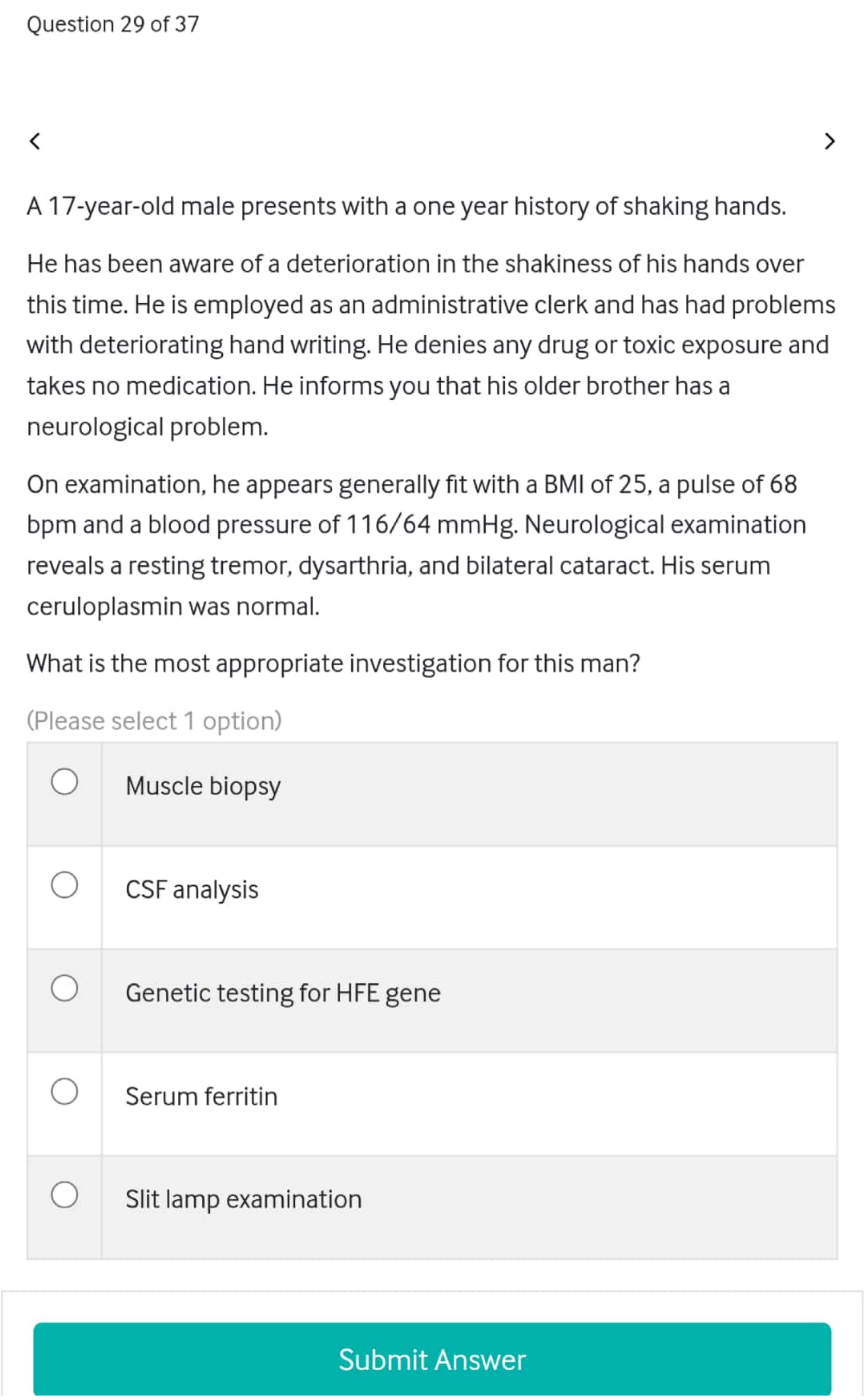
There are three products used, beta-interferon 1a (Avonex and Rebif), both of which are licensed for relapsing-remitting MS and beta-interferon 1b (Betaferon), licensed for both relapsing remitting and for secondary progressive forms of MS.

Acute management with steroids (oral or intravenously) is unwarranted given the nature of the symptoms and the fact that they are improving.

CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) is a differential diagnosis for transient neurological symptoms associated with multiple white matter changes, but would not cause spinal cord changes.

Mitoxantrone would not be first line treatment for relapsing remitting MS, and is reserved for high frequency relapse rates unresponsive to beta-interferon.

Next Question



A 17-year-old male presents with a one year history of shaking hands.

He has been aware of a deterioration in the shakiness of his hands over this time. He is employed as an administrative clerk and has had problems with deteriorating hand writing. He denies any drug or toxic exposure and takes no medication. He informs you that his older brother has a neurological problem.

On examination, he appears generally fit with a BMI of 25, a pulse of 68 bpm and a blood pressure of 116/64 mmHg. Neurological examination reveals a resting tremor, dysarthria, and bilateral cataract. His serum ceruloplasmin was normal.

What is the most appropriate investigation for this man?

(Please select 1 option)

○ Muscle biopsy

○ CSF analysis

○ Genetic testing for HFE gene

○ Serum ferritin

○ Slit lamp examination

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● Slit lamp examination **Correct**

Key Learning Points

Neurology

- Wilsons disease presents with KF rings and have abnormal brain MR findings.

Explanation

Ceruloplasmin is normal in approximately 5% of cases of Wilson's disease.

However, the features of this case would point to this disorder particularly with the family history.

Wilson's disease can cause resting, postural, cerebellar, and a wing beating type of tremor. Wilson's disease can cause proximal renal tubular acidosis (RTA) type 2 which is almost always non-significant; so sending for serum electrolytes (Na, K, Cl, and HCO_3) and looking for a normal anion gap metabolic acidosis are unnecessary.

A slit lamp examination will detect Kayser-Fleischer corneal rings in approximately 98% of untreated cases and the sunflower cataract will be more obvious.

Serum copper, 24 hours urinary copper, and liver biopsy were not mentioned in the options, but are the corner stones in the diagnosis.

MRI studies have identified focal abnormalities in the white matter, pons, and deep cerebellar nuclei.

Next Question

A 65-year-old man was admitted to the Emergency department with confusional state.

His wife, who accompanied him, said he had been behaving bizarrely for the past two days, fumbling with his clothes and making strange swallowing movements. Prior to this, he had had several twitching episodes of his right hand and periods of speech impairment. He had not complained of headache or any visual disturbances. His past medical history included type II diabetes; hypertension and longstanding partial epilepsy.

His regular prescribed medications included metformin 1 g daily, bendroflumethiazide 2.5 mg daily, phenytoin 300 mg daily and carbamazepine 1.2 g.

He was a smoker of 25 cigarettes per day and drank 10 units of alcohol per week.

On examination he appeared confused with a Glasgow coma scale of 13/15 (M 6 E 4 V3). His blood pressure was 146/89 mmHg, oxygen saturation 96% on room air, pulse was 87/min and regular and temperature was 37.5°C.

There was no neck stiffness or photophobia. He exhibited subtle nystagmus, however, eye movements were normal. The rest of the cranial nerves were intact. Examination of the peripheral nervous system revealed some intermittent twitching of the right hand with increased tone and brisk reflexes in the right upper limb.

Chest and cardiovascular examination revealed left basal crackles.

Investigations showed:

Haemoglobin	136 g/L	(130-180)
White cell count	15 $\times 10^9$/L	(4-11)
Platelets	285 $\times 10^9$/L	(150-400)
Serum sodium	135 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	5.6 mmol/L	(2.5-7.5)
Serum creatinine	98 µmol/L	(60-110)
Serum corrected calcium	2.5 mmol/L	(2.2-2.6)
Plasma glucose	4.6 mmol/L	(3.0-6.0)
C reactive protein	8 mg/L	(<5)
Phenytoin level	42 mol/L	(Therapeutic range 40-80)
Carbamazepine level	37 mol/L	(Therapeutic range 34-51)

A CT scan of the brain showed multiple periventricular ischaemic white matter changes.

Given the history and clinical findings so far, how would you manage this patient?

(Please select 1 option)

○ Commence on intravenous thiamine and vitamin B co-strong

○ Refer to psychiatry

○ Commence on intravenous lorazepam and oral amoxicillin

○ Commence on intravenous aciclovir 10 mg/kg/day

○ Commence on aspirin 300 mg daily

Submit Answer

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○ Commence on intravenous lorazepam and oral amoxicillin
This is the correct answer

○ Commence on intravenous aciclovir 10 mg/kg/day

○ Commence on aspirin 300 mg daily

Key Learning Points

Neurology

- Differential diagnoses of non-convulsive status include: metabolic encephalopathy, transient ischaemia, migraine, infection, psychiatric causes.

Explanation

The clinical history would be in keeping with non-convulsive partial status, and seizures possibly triggered by infection.

He has long-standing partial epilepsy on multiple therapies and there is a clear period prior to the confusional state of simple partial motor seizures.

In addition, it is likely he has an underlying chest infection which may have additionally triggered status.

Differential diagnoses of non-convulsive status include:

- toxic or metabolic encephalopathies
- transient cerebral ischaemia
- migraine
- infection, or
- psychiatric causes.

This patient requires ambulatory EEG monitoring, IV lorazepam and commencement of oral antibiotics.

CT Head Imaging: "Casues of periventricular white matter (PWM) lesions include normal senescent changes (then they are called UBO's, for unidentified bright objects), small strokes, and disorders related to multiple sclerosis (MS). PWM are correlated with vitamen B6 (pyridoxine) deficiency."

A 32-year-old woman was seen in the neurology clinic with a four day history of flu-like symptoms, double vision and facial weakness.

She complained of double vision on looking to the right and was aware that she could not fully close her eyes. She had come back six weeks ago from a holiday in the New Forest, where she had been camping with a friend. She had a past medical history of type I diabetes diagnosed at the age of 8 years and took regular insulin injections.

On examination she appeared alert and orientated. She complained of arthralgia in her hands and knees. Her blood pressure was 130/75 mmHg, pulse was 70/min and regular, temperature was 37.4°C and blood glucose monitoring was 4.7 mmol/L. There was no evidence of rash or any neck stiffness.

On cranial nerve examination, fundoscopy was normal and both pupils were equal and reactive to light. On testing ocular movements, there was oculoparesis of the right lateral rectus. There was also bilateral facial weakness with bilateral Bell's phenomenon. Examination of the peripheral nervous system did not reveal any abnormalities.

A lumbar puncture was performed and yielded the following data:

Opening pressure	13 cmH_2O	(5-18)
CSF protein	1.3 g/L	(0.15-0.45)
CSF white cell count	120 per ml	(<5)
CSF white cell differential	90% lymphocytes	-
CSF red cell count	4 per ml	(<5)
CSF glucose	3.9 mmol/L	(3.3-4.4)
CSF oligoclonal bands	Present	-
Serum glucose	5.7 mmol/L	(3.0-6.0)
Serum oligoclonal bands	Negative	-

A MRI scan of the brain showed multiple periventricular white matter lesions.

Given the above history and clinical findings, how would you manage this patient?

(Please select 1 option)

○ Commence on aciclovir 10 mg/kg intravenously TDS and cefotaxime 2 g tds

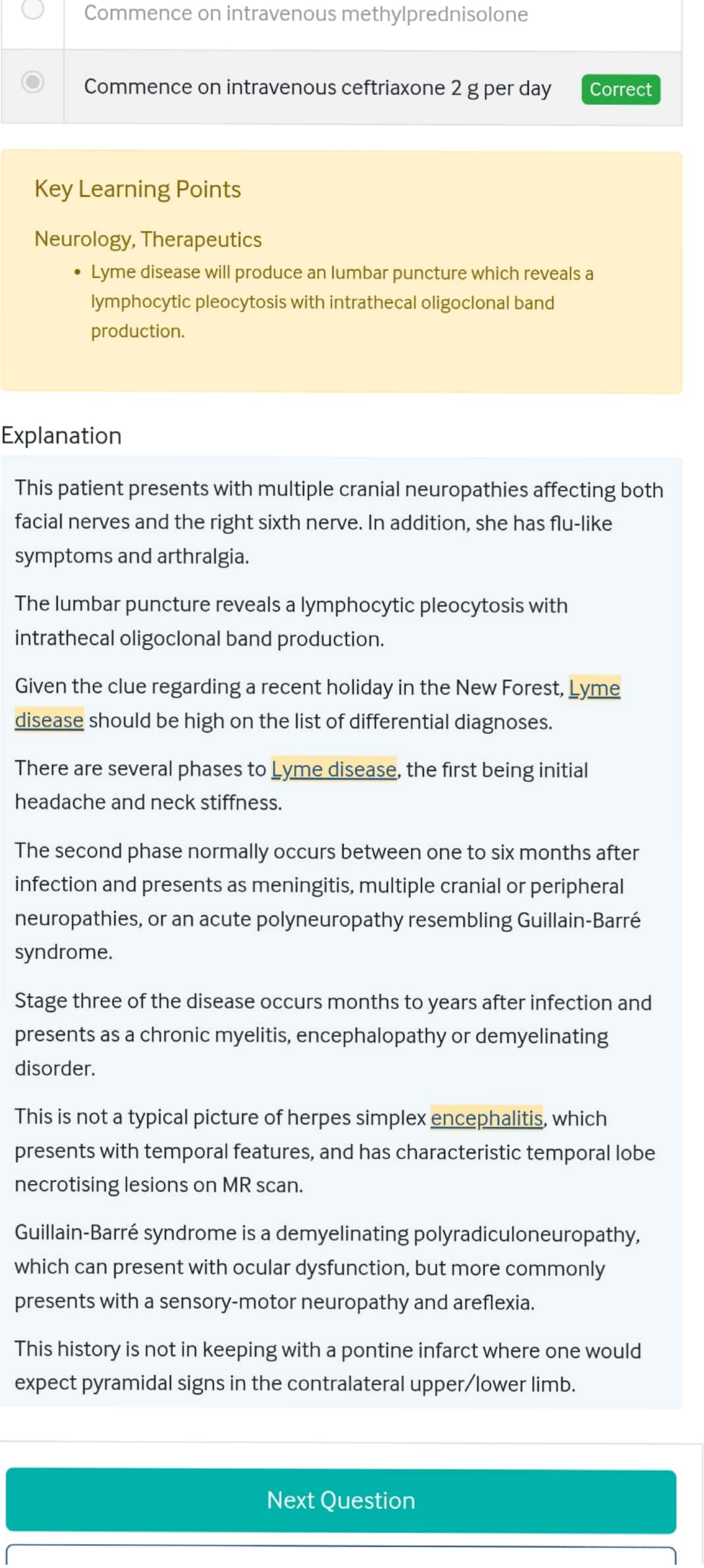
○ Commence on intravenous immunoglobulin

○ Start on aspirin and a statin and optimise her diabetes

○ Commence on intravenous methylprednisolone

○ Commence on intravenous ceftriaxone 2 g per day

Submit Answer



○ Commence on intravenous methylprednisolone

● Commence on intravenous ceftriaxone 2 g per day

Key Learning Points

Neurology, Therapeutics

- Lyme disease will produce an lumbar puncture which reveals a lymphocytic pleocytosis with intrathecal oligoclonal band production.

Explanation

This patient presents with multiple cranial neuropathies affecting both facial nerves and the right sixth nerve. In addition, she has flu-like symptoms and arthralgia.

The lumbar puncture reveals a lymphocytic pleocytosis with intrathecal oligoclonal band production.

Given the clue regarding a recent holiday in the New Forest, Lyme disease should be high on the list of differential diagnoses.

There are several phases to Lyme disease, the first being initial headache and neck stiffness.

The second phase normally occurs between one to six months after infection and presents as meningitis, multiple cranial or peripheral neuropathies, or an acute polyneuropathy resembling Guillain-Barré syndrome.

Stage three of the disease occurs months to years after infection and presents as a chronic myelitis, encephalopathy or demyelinating disorder.

This is not a typical picture of herpes simplex encephalitis, which presents with temporal features, and has characteristic temporal lobe necrotising lesions on MR scan.

Guillain-Barré syndrome is a demyelinating polyradiculoneuropathy, which can present with ocular dysfunction, but more commonly presents with a sensory-motor neuropathy and areflexia.

This history is not in keeping with a pontine infarct where one would expect pyramidal signs in the contralateral upper/lower limb.

A 52-year-old right-handed man was admitted to hospital having had several generalised tonic clonic seizures whilst at home.

Prior to having the seizures he had complained of generalised headache associated with nausea. His wife who accompanied him in hospital said that he had been having some difficulties using his right hand and on occasions had difficulties with expressive speech in the last two months. Otherwise he had not complained of any other symptoms and there was no history of head injury.

He had recently been to see his GP complaining of a productive cough and was awaiting pulmonary function tests. Otherwise he had no known past medical history and did not take any regular medication. He was a smoker of 40 cigarettes per day and drank 12 units of alcohol per week.

On examination he was drowsy but rousable with a Glasgow coma scale of 13/15 (M 6 E 3 V4). His blood pressure was 150/90 mmHg, pulse was 67/min and regular and temperature was 35.8°C.

Chest examination revealed bilateral expiratory wheeze. Cardiovascular and gastrointestinal examinations were entirely normal. Neurological examination revealed some expressive dysphasia with a right homonymous hemianopia. Pupillary responses and the rest of the cranial nerve examination was normal. Peripheral nervous system examination revealed a right sided hemiparesis with increased tone, pyramidal weakness and pathologically brisk reflexes. Both plantar responses were extensor.

Investigations revealed:

Haemoglobin	182 g/L	(130-180)
White cell count	11 ×10^9/L	(4-11)
Platelets	450 ×10^9/L	(150-400)
C reactive protein	10 mg/L	(<10)
Sodium	134 mmol/L	(137-144)
Urea	6.7 mmol/L	(3.5-4.9)
Potassium	4.1 mmol/L	(3.5-4.9)
Creatinine	88 µmol/L	(60-110)
Glucose	5.6 mmol/L	(3.0-6.0)

The chest x ray was normal.

A lumbar puncture revealed:

CSF opening pressure	22cmH20	(4-18)
CSF protein	1.1g/L	(0.15-0.45)
CSF white cell count	20 cells per ml (90% lymphocytes)	
CSF glucose	3.6 mmol/L	(3.3-4.4)

A gadolinium enhanced MRI brain scan showed a poorly demarcated ring-enhancing lesion within the left fronto-parietal area with some mass effect.

Given the above clinical account and investigations, what is the most likely diagnosis in this patient?

(Please select 1 option)

○ Metastatic lung carcinoma

○ Multiple meningiomas

○ Glioblastoma multiforme

○ Multiple cerebral abscesses

○ Diffuse infiltrative primary CNS lymphoma

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○ Glioblastoma multiforme **This is the correct answer**

○ Multiple cerebral abscesses

○ Diffuse infiltrative primary CNS lymphoma

Key Learning Points

Neurology

- Meningiomas are confined to the meninges, whilst primary CNS lymphomas have a predilection to periventricular white matter.

Explanation

This patient presents with headaches, seizures and focal neurological signs and symptoms.

He has poorly demarcated non-homogenous ring enhancing lesion on MRI imaging which appear to extend to the meninges. He has a mild lymphocytic pleocytosis and markedly raised cerebrospinal fluid (CSF) protein associated with elevated CSF opening pressure.

Other than raised haemoglobin which may reflect polycythaemia secondary to chronic bronchitis and smoking, his blood tests appear normal.

The most likely diagnosis is glioblastoma multiforme.

Metastatic lung carcinoma is unlikely in view of a normal CXR.

Cerebral abscesses are very unlikely in view of normal inflammatory markers, CSF white cell count and normal glucose level and apyrexia.

Meningiomas and primary cerebral lymphomas are typically homogenous and well demarcated lesions.

Meningiomas are confined to the meninges, whilst primary CNS lymphomas have a predilection to periventricular white matter.

Next Question

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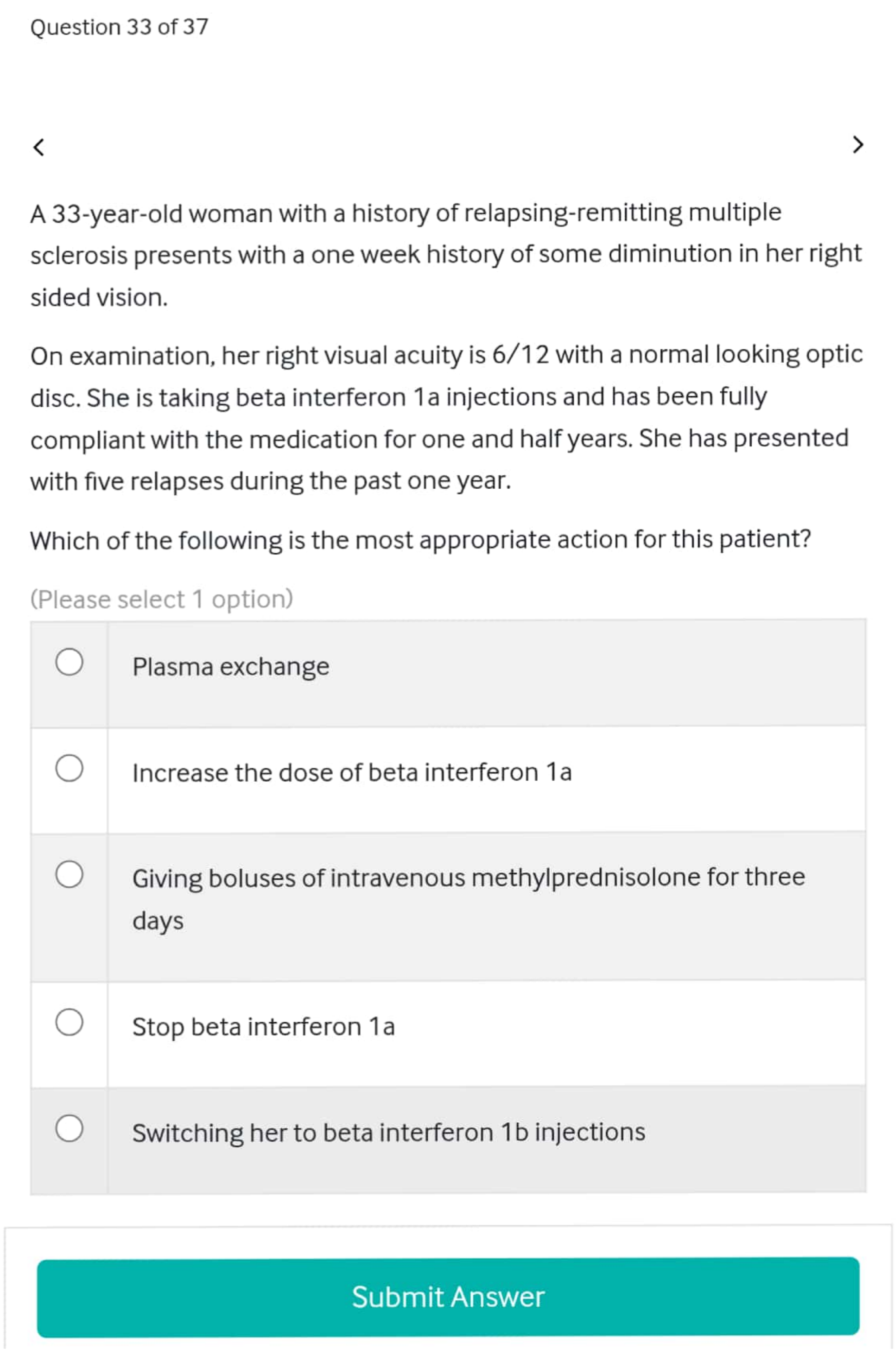

Question 33 of 37

A 33-year-old woman with a history of relapsing-remitting multiple sclerosis presents with a one week history of some diminution in her right sided vision.

On examination, her right visual acuity is 6/12 with a normal looking optic disc. She is taking beta interferon 1a injections and has been fully compliant with the medication for one and half years. She has presented with five relapses during the past one year.

Which of the following is the most appropriate action for this patient?

(Please select 1 option)

- ○ Plasma exchange
- ○ Increase the dose of beta interferon 1a
- ○ Giving boluses of intravenous methylprednisolone for three days
- ○ Stop beta interferon 1a
- ○ Switching her to beta interferon 1b injections

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○ Stop beta interferon 1a This is the correct answer

○ Switching her to beta interferon 1b injections

Key Learning Points

Neurology

- Only life threatening or function changes in MS require high dose pulsed methylprednisolone.

Explanation

This woman with a history of relapsing-remitting multiple sclerosis is experiencing a mild non-visual threatening attack of optic neuritis.

These attacks do not need any specific therapy; only function- or life-threatening relapses should be treated by high doses of bolus methylprednisolone.

Three or more relapses per year preclude any continuation of the interferons or glatiramer acetate injections (as the objective behind using them is to reduce relapse frequency).

Plasma exchange has a place in severe function- or life-threatening relapses not responding to conventional treatment.

Some patients with RRMS have disease activity that is refractory to initial disease-modifying therapy with interferon beta (IFNB) drugs or glatiramer acetate. In this situation,as there is limited evidence available for each of these drugs, it is recommended to review therapy. Review may include changing the agent entirely to a drug with a different mechanism of action.

Next Question

A 42-year-old woman presented to her GP complaining of visual impairment. She had noticed for some time that she had been bumping into things particularly at furniture height affecting both sides.

Over the last two weeks, she had developed worsening generalised headaches and had recently had two complex partial seizures manifest as a vague epigastric sensation followed by altered awareness and fumbling with clothes lasting several minutes.

She had a past history of partial epilepsy diagnosed at 20 years of age and had previously been treated with vigabatrin. Her current medication was lamotrigine 400 mg/day. She was a smoker of 10 cigarettes/day and drank 8 units of alcohol per week.

On examination, her blood pressure was 136/78 mmHg with no postural drop, pulse was 89/minute and regular and heart sounds were normal. On cranial nerve examination, fundoscopy appeared normal, pupillary responses were intact however there was evidence of a marked bitemporal inferior quadrantinopia. There was also some paraesthesia affecting the left ophthalmic branch. Ocular movements were full. There were no abnormalities detected in the peripheral nervous system.

Investigations showed:

Haemoglobin	134 g/L	(115 - 165)
White cell count	6.2 $\times 10^9$/L	(4 - 11)
Platelets	199 $\times 10^9$/L	(150 - 400)
Serum sodium	136 mmol/L	(137 - 144)
Serum potassium	4.2 mmol/L	(3.5 - 4.9)
Serum urea	6.7 mmol/L	(2.5 - 7.5)
Serum creatinine	110 µmol/L	(60 - 110)
Prolactin	1100 U/L	(<600U/L)

Given the above clinical account what is the likely diagnosis?

(Please select 1 option)

- ○ Vigabatrin-induced visual field defect
- ○ Parietal tumour
- ○ Craniopharyngioma
- ○ Pituitary adenoma
- ○ Glaucoma

○ Craniopharyngioma **This is the correct answer**

○ Pituitary adenoma

○ Glaucoma

Key Learning Points

Neurology

- Compression of the optic chiasm from above causes a bilateral inferior quadrantinopia.

Explanation

The most likely diagnosis is a craniopharyngioma compressing the chiasm from above leading to a bilateral inferior quadrantinopia.

There is probably some cavernous sinus extension given the trigeminal nerve involvement and also compression of the pituitary stalk, leading to overproduction of prolactin. The history also suggests medial temporal lobe involvement manifest by two recent temporal lobe seizures.

A pituitary adenoma is more likely to cause a superior bitemporal field defect initially, and one might expect to see signs or symptoms of pituitary dysfunction.

A parietal tumour would cause a unilateral inferior quandrantinopic field defect.

Vigabatrin-associated field defects are typically nasal more so than temporal, and would not explain the additional signs and symptoms in this patient.

Glaucoma producing a marked field defect would be evident on fundoscopy.

<   >

A 63-year-old man with a history of type 2 diabetes and hypertension is admitted to the Emergency Department some five hours after suffering weakness affecting the right hand side of his face and his right arm.

On initial assessment, you confirm the obvious weakness, and it is apparent the patient has suffered an acute stroke.

In this situation systolic blood pressure above what level would require acute treatment?

(Please select 1 option)

○ 160 mmHg

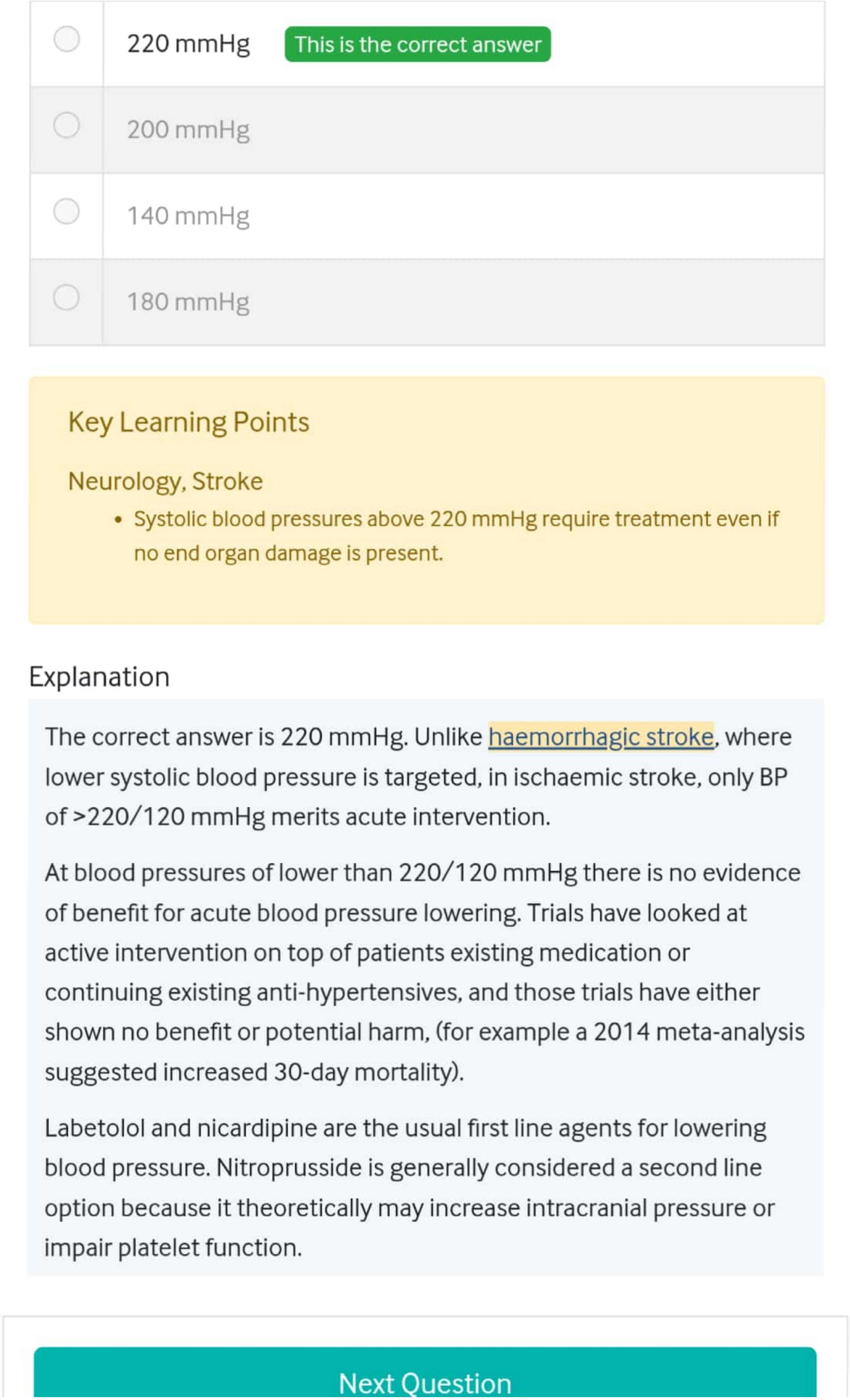
○ 220 mmHg

○ 200 mmHg

○ 140 mmHg

○ 180 mmHg

Submit Answer



○ 220 mmHg This is the correct answer

○ 200 mmHg

○ 140 mmHg

○ 180 mmHg

Key Learning Points

Neurology, Stroke

- Systolic blood pressures above 220 mmHg require treatment even if no end organ damage is present.

Explanation

The correct answer is 220 mmHg. Unlike haemorrhagic stroke, where lower systolic blood pressure is targeted, in ischaemic stroke, only BP of >220/120 mmHg merits acute intervention.

At blood pressures of lower than 220/120 mmHg there is no evidence of benefit for acute blood pressure lowering. Trials have looked at active intervention on top of patients existing medication or continuing existing anti-hypertensives, and those trials have either shown no benefit or potential harm, (for example a 2014 meta-analysis suggested increased 30-day mortality).

Labetolol and nicardipine are the usual first line agents for lowering blood pressure. Nitroprusside is generally considered a second line option because it theoretically may increase intracranial pressure or impair platelet function.

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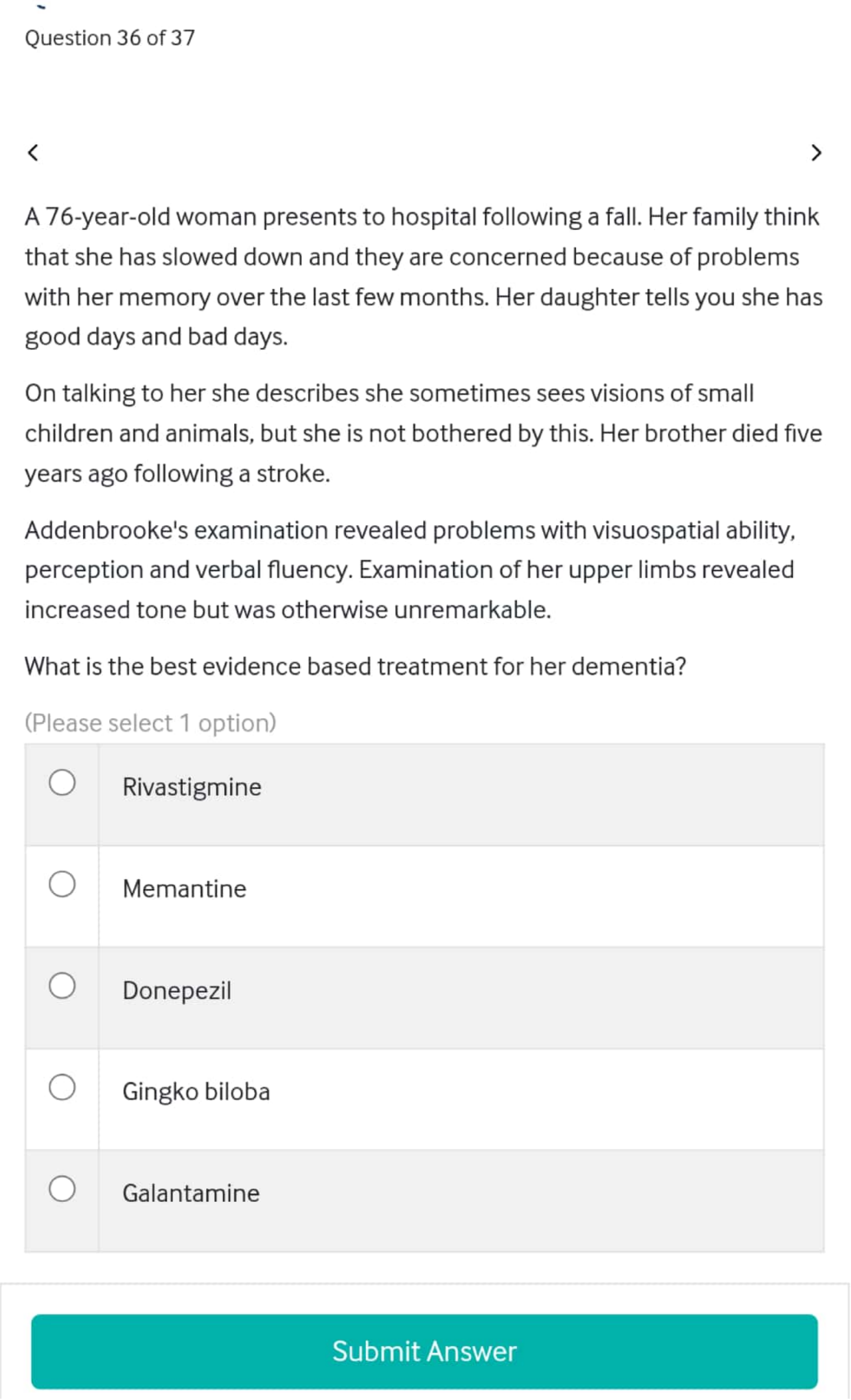


Question 36 of 37

A 76-year-old woman presents to hospital following a fall. Her family think that she has slowed down and they are concerned because of problems with her memory over the last few months. Her daughter tells you she has good days and bad days.

On talking to her she describes she sometimes sees visions of small children and animals, but she is not bothered by this. Her brother died five years ago following a stroke.

Addenbrooke's examination revealed problems with visuospatial ability, perception and verbal fluency. Examination of her upper limbs revealed increased tone but was otherwise unremarkable.

What is the best evidence based treatment for her dementia?

(Please select 1 option)

- ○ Rivastigmine
- ○ Memantine
- ○ Donepezil
- ○ Gingko biloba
- ○ Galantamine

○ Rivastigmine **This is the correct answer**

○ Memantine

● Donepezil **Incorrect answer selected**

○ Gingko biloba

○ Galantamine

Key Learning Points

Neurology

- Rivastigmine is effective in treating cognitive decline in dementia with Lewy bodies. It may be effective in reducing anxiety, apathy and hallucinations.

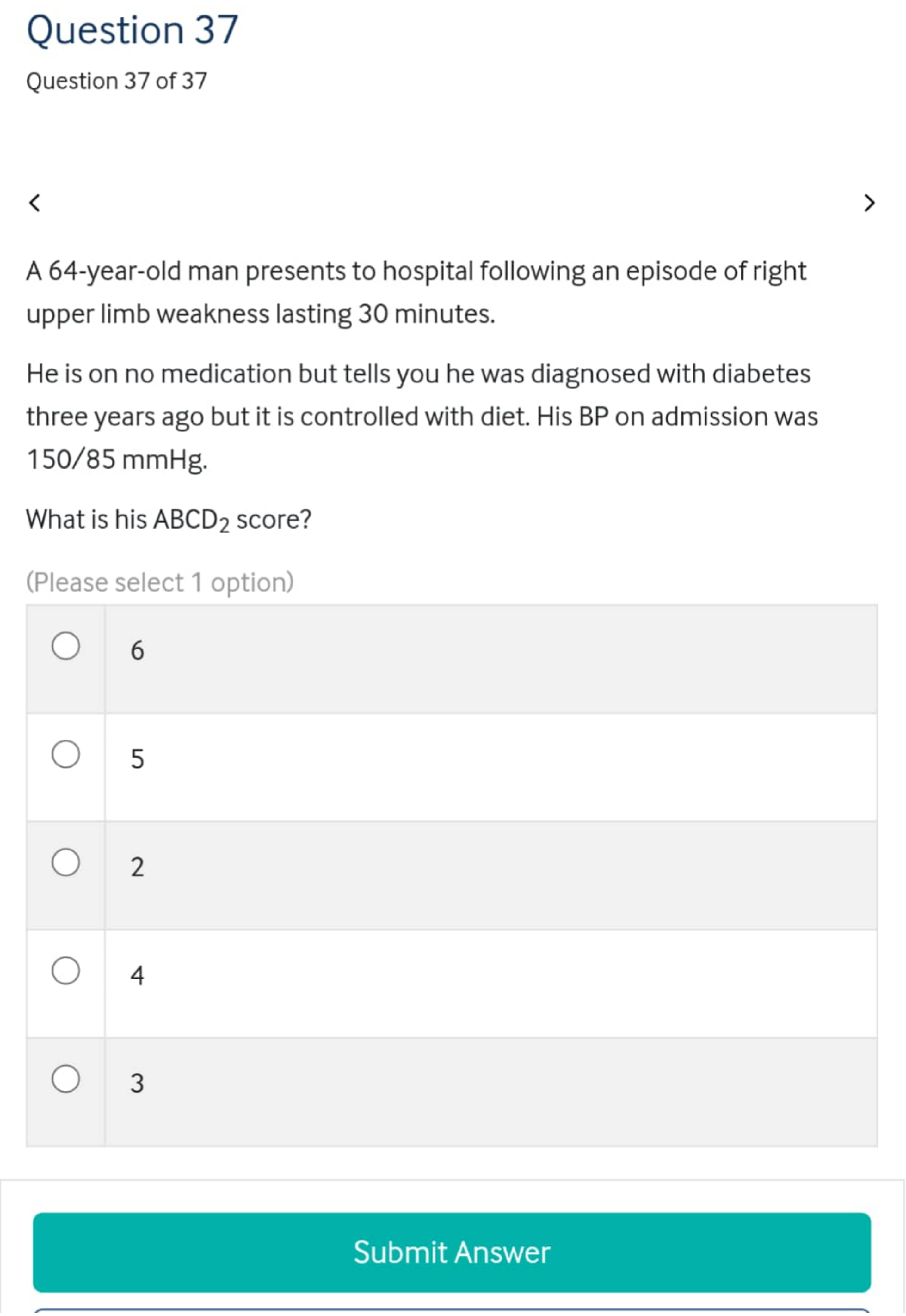
Explanation

Rivastigmine is effective in treating cognitive decline in dementia with Lewy bodies. It may be effective in reducing anxiety, apathy and hallucinations; it has been shown to be better than placebo in LBD and for hallucinations.

Donepezil and galantamine are used in the treatment of Alzheimer's dementia.

Gingko biloba is a herbal medication which has not been proven to be of benefit in treatment or prevention of dementia.

Memantine is used only in moderately severe to severe Alzheimer's dementia. It is usually started at 5mg and increased in steps of 5mg every week until 20mg dose.



Question 37

Question 37 of 37

A 64-year-old man presents to hospital following an episode of right upper limb weakness lasting 30 minutes.

He is on no medication but tells you he was diagnosed with diabetes three years ago but it is controlled with diet. His BP on admission was 150/85 mmHg.

What is his $ABCD_2$ score?

(Please select 1 option)

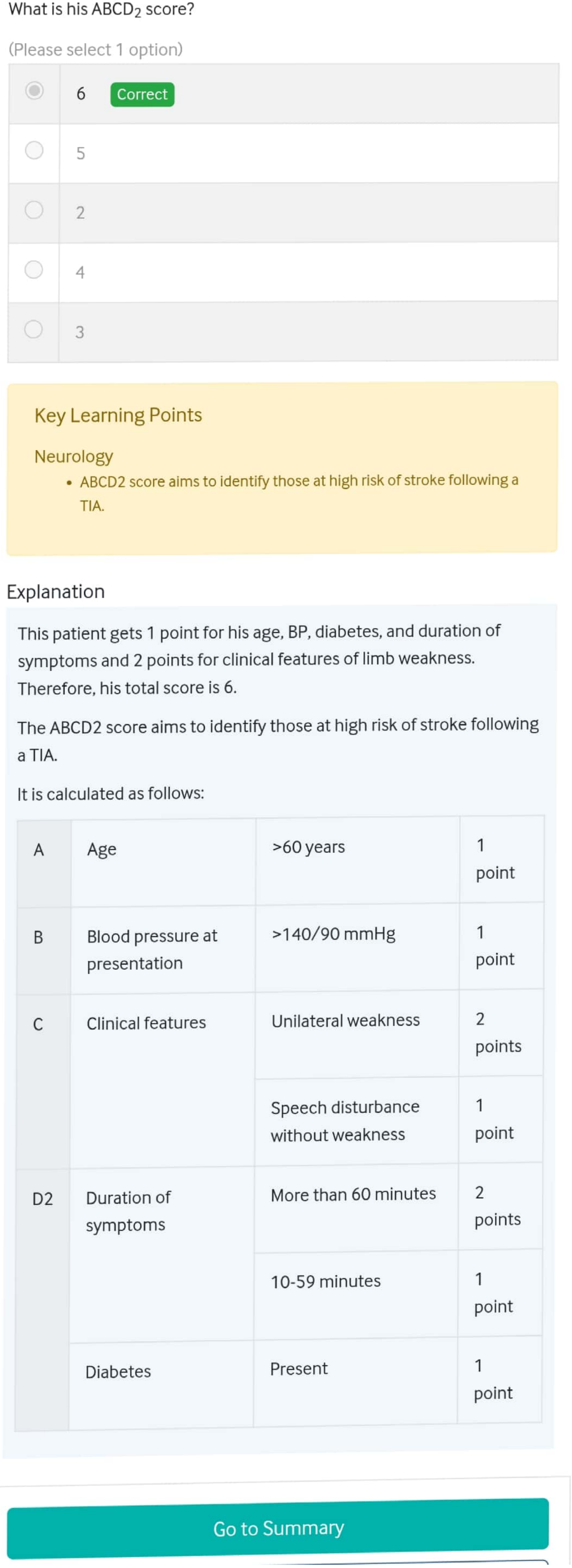
○ 6

○ 5

○ 2

○ 4

○ 3



What is his $ABCD_2$ score?

(Please select 1 option)

● 6 Correct

○ 5

○ 2

○ 4

○ 3

Key Learning Points

Neurology

- ABCD2 score aims to identify those at high risk of stroke following a TIA.

Explanation

This patient gets 1 point for his age, BP, diabetes, and duration of symptoms and 2 points for clinical features of limb weakness. Therefore, his total score is 6.

The ABCD2 score aims to identify those at high risk of stroke following a TIA.

It is calculated as follows:

A	Age	>60 years	1 point
B	Blood pressure at presentation	>140/90 mmHg	1 point
C	Clinical features	Unilateral weakness	2 points
		Speech disturbance without weakness	1 point
D2	Duration of symptoms	More than 60 minutes	2 points
		10-59 minutes	1 point
	Diabetes	Present	1 point

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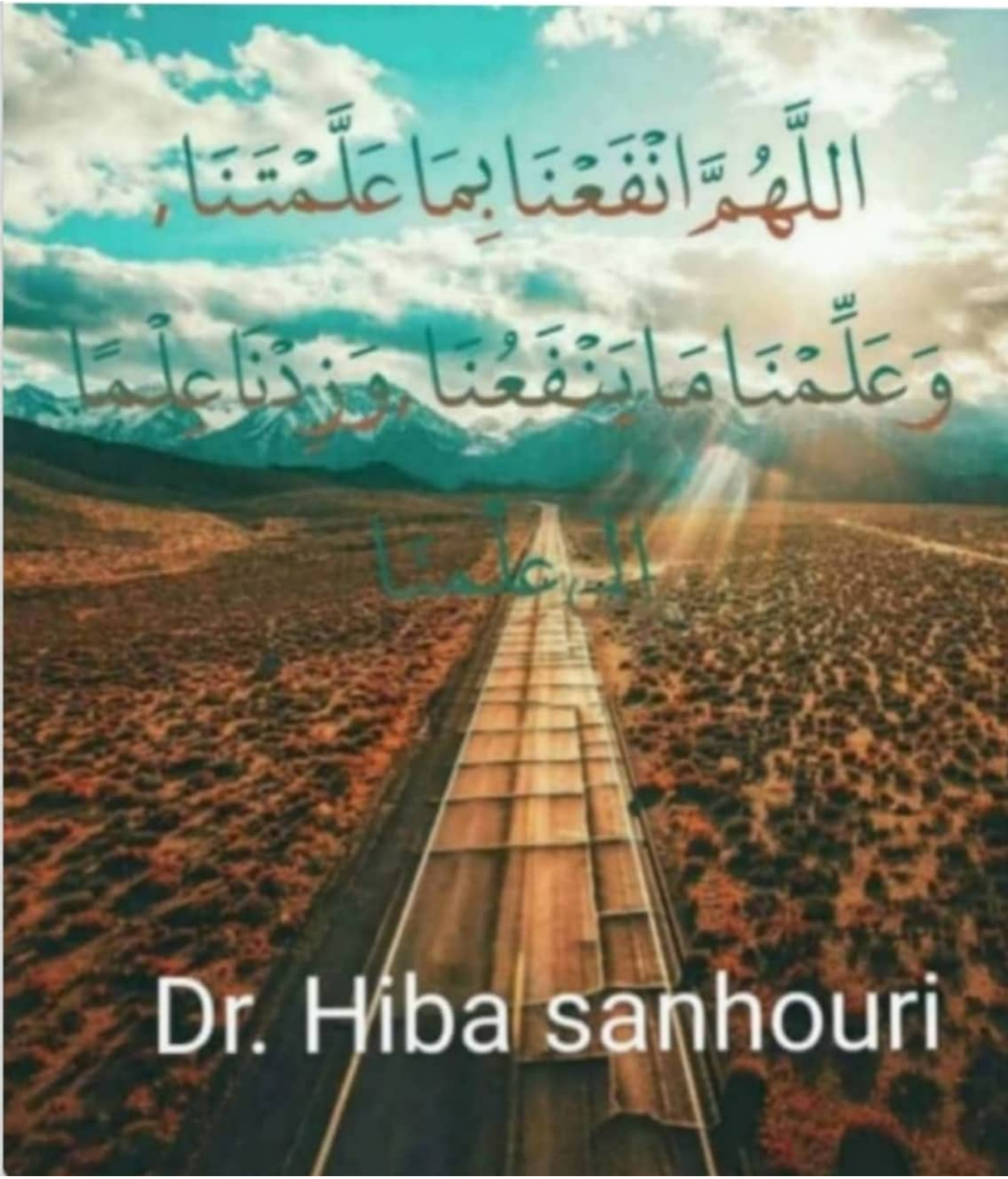
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وَعَلِّمْنَا مَا يَنْفَعُنَا وَزِدْنَا عِلْمًا
Dr. Hiba sanhouri